The Journal
OF THE
AMERICAN ASSOCIATION
OF NURSE ANESTHETISTS

CONTENTS

OPINION REVIEW
The Question of Accreditation 144

EDITORIAL
It's Every Member's Annual Meeting 149

INTRAVENOUS USE OF PROCAINE 150
Martha M. Veilleux, R.N.

PREOPERATIVE AND POSTOPERATIVE FLUID THERAPY 155
William J. Pendergrast, M.D.

POSTOPERATIVE URINARY SUPPRESSION DUE TO ANOXIA 160
Ormond S. Culp, M.D.

OXYGEN LACK, CARBON DIOXIDE EXCESS, AND ASPHYXIA 168
John S. Gray, Ph.D., M.D.

BALANCED ANESTHESIA AND SUPPORTIVE THERAPY 174
John S. Lundy, M.D., John Waugh, M.D., and R. C. Adams, M.D.

NOTES 194

LEGISLATION 196

THE NEWS 198

ABSTRACTS 209

BOOK REVIEWS 213

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1. Goodman, L., and Gilman, A.: *The Pharmacological Basis of Therapeutics*, New York, The Macmillan Co., 1947, p. 58.

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OPINION REVIEW

THE QUESTION OF ACCREDITATION

After many years of progress forward and upward to the achievement of the present high level of skill and professional influence, the field of nurse anesthesia is approaching a crisis that can be resolved in either of two ways: through the consolidation of gains for which we have fought so diligently and successfully, or through permitting a lapse into decadence that would eventually reduce our status to that of technician assistants to physician administrators of anesthetics.

You know what that would eventually do to the independence and position of the nurse anesthetist as we know her today. Recognizing the danger in this policy if it were permitted to become entrenched, our national association with the co-operation of the American Hospital Association is planning a vigorous program to defend our schools against such an attack.

Our association's plan is to develop a program under which schools of anesthesia for nurses will be nationally accredited by the American Association of Nurse Anesthetists. This would enable the Association to demand and to see that the schools maintain appropriate standards of teaching both as to quality and to quantity. Under such a plan the schools would of necessity have to give proper education and training to nurse anesthetists if the schools were to be certified and if the graduates were to be accepted by hospitals generally—no matter who headed the schools.

This comprehensive and far reaching project naturally will require a considerable sum of money, first, to employ competent professional technical assistance and, second, to employ a permanent educational director to visit every school to make certain that it maintains high standards of training, whether or not a physician anesthetist is the director of the school.

To raise the funds needed to carry this constructive project forward successfully will necessitate raising the dues of the members of the A.A.N.A. substantially, but the cost will be modest when considered in the light of the future of on-coming nurse anesthetists, as well as of nurses already in the field.—HELEN LAMB, R.N., Chairman, Advisory to the Approval Committee, St. Louis.

To retain the place we have built for ourselves in the hospital world and to assure ourselves that schools of anesthesia for nurses will send out only well trained, efficient nurse anesthetists, it is mandatory that we institute some form of program for the accreditation of schools. Over the years untold time and work have been spent by our most capable members, acting on various committees, to build the foundation for the project. It is recommended that every member study the A.A.N.A. NEWS BULLETIN and the JOURNAL carefully and learn the importance of the program. The plan, when accepted, must be implemented financially. Every voting member

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should study the "wide view" of the benefit to the group and not the "narrow view" of "I can't afford any more dues." The plan to be presented at the annual meeting in September has already cost dearly in time and work by very busy people as well as in money. I believe it will be worth a few dollars a year to every member to have the accreditation program put into operation.—MINNIE V. HAAS, R.N., Fort Worth, Tex.

For several years I have heard members of the A.A.N.A. complain that there are some of our group who, although they have passed the qualifying examination, are not really equipped to fill a position as anesthetist capably. Not only does this complaint come from those working with these women, but it comes from the ill equipped themselves.

The A.A.N.A. has tried to see to it that all schools that have been recognized do meet certain standards. These standards, of necessity, have been accepted almost solely by trusting the integrity of the schools applying for recognition. We have not truly had a program of standards, nor have we had the personnel for checking the veracity of the applicant.

It is grossly unfair for a student to spend valuable time and money to become an able anesthetist and to depart with only half knowledge in theory or in practical experience at the end of her course of study. It is also grossly unfair that there are schools graduating students in such a manner as to pass the qualifying examination but so unfit to fill a position as a nurse anesthetist as to discredit those who are capable.

There is no slander in the statement that there are hospitals operating schools of anesthesia purely to fill their own needs for anesthesia service. It is only too tragically true. This statement is not meant to be uncharitable. We full well realize the acute shortage of anesthetists of any sort, and the A.H.A. has been most co-operative in helping us to produce a program for the approval of schools. We now have that program. What we lack are the funds for following through.

Therefore, the members of the A.A.N.A. MUST come forth with a vote for a BIG increase in dues—our only real source of income. Heave ho, let's launch that ship: Approval of Schools Program!—VERNA E. BEAN, R.N., Lexington, Ky.

In my opinion there is a great need for an approval program for our schools. At the present time the only way a school can be judged is by the standing of its students who take the qualifying examination. Our aim should be to develop schools where the students not only receive the proper theoretical instruction but also are trained to apply this knowledge to the problems encountered in anesthesia. Therefore, we should have a program by which our schools may be judged from the practical as well as the theoretical standpoint.

Such a program would involve a marked increase in our expenditures, which would have to be met by an increase in dues. I feel that the results would justify the cost of the project.—LILLIAN G. BAIRD, R.N., Ann Arbor, Mich.

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The Journal of the American Association of Nurse Anesthetists

VOLUME XVIII AUGUST, 1950 NUMBER THREE

IT'S EVERY MEMBER'S ANNUAL MEETING

"'The time has come,' the Walrus said,
To talk of many things:"
—Lewis Carroll

Yes, the time for our annual meeting is drawing near, and we wish to urge you to make every effort to attend that meeting. It affords the opportunity to "talk over" our mutual problems.

Our association has reached a point in its development where it must branch out and meet the challenge brought about by the scope of its activities. You should decide if and how we are to meet our responsibilities. Specifically, our association should sponsor its school or be in a position to offer assistance to those schools if their training programs are inadequate. These training centers are the life-line of the Association in raising the standards of the nurse anesthetist.

Other professional organizations have put into operation their plans for accreditation. We must do likewise in order to justify our existence as a professional organization.

We further suggest that each Assembly or Section plan to send a representative to this meeting. This is an occasion for an exchange of ideas that should be to the advantage of all concerned—MYRA VAN ARSDALE, R.N., President.

OFFICIAL CALL

As provided for in the Bylaws of this association, and at the direction of Myra Van Arsdale, president, we hereby issue this official call to the members of the annual meeting to be held in Atlantic City, September 18-21, 1950, in Room A of Convention Hall. The annual business session will be held on Tuesday, September 19.

Accomplished at the Executive Offices, 22 East Division St., Chicago 10, Ill., this 28th day of June 1950.

(Signed) FLORENCE A. MCQUILLEN
Executive Director

INTRAVENOUS USE OF PROCAINE

Martha M. Veilleux, R.N.*
Detroit

The discovery that procaine may be injected into a vein with comparatively little risk was first made by Bier in 1909. This technic, however, enjoyed little popularity until 1940 when Lundy[1] first used it successfully in the treatment of pruritus of jaundice.

In several fields of clinical investigation workers have found that procaine is an extremely versatile therapeutic drug as well as a valuable local anesthetic agent. Its administration by the intravenous route relieves not only pain but other symptoms in many unrelated conditions.

Procaine is a *p*-aminobenzoyldiethylamino-ethanol, a synthetic product, generally used in the form of the hydrochloride, which occurs as colorless crystals or a white crystalline powder.[2] It is soluble in water, melts at 156 C., is stable, and does not decompose at the temperature of boiling water. In solution it is precipitated by alkaloid reagents.

Small doses produce a slight increase in blood pressure and stimulate respiration, while large doses cause an abrupt decrease in blood pressure. If a lethal dose is administered slowly, death is due to asphyxia from paralysis of the respiratory center. Hirschel[3] estimated that procaine administered intravenously is sixteen times as toxic as when it is administered subcutaneously. The tolerance of an adult patient under anesthesia has been found to be 1 Gm. of the drug.

Studies by Hatcher and Eggleston[4] demonstrated the relative safety of intravenous injection of procaine in dilute solution and its rapid destruction. In the blood stream of rabbits and of human beings it disappears within thirty minutes of injection. In the human blood procaine is hydrolyzed by an esterase, some of the acid formed then undergoing acetylation by the liver. This esterase, or enzyme, appears to be contained in blood plasma and is probably formed in the liver. It can be inhibited by the administration of neostigmine, after which procaine toxicity is increased in mice, rats, and dogs. In view of these results the simultaneous administration of neostigmine and procaine would appear to involve some additional hazard.

In the preparation of procaine solution, as a rule, 1 Gm. of procaine hydrochloride crystals is dissolved in 1 L. normal saline solution to make a 0.1 per cent concentration. Infusion rates vary

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Read before the Michigan Association of Nurse Anesthetists, Detroit, Nov. 12, 1949.
*Department of Anesthesia, The Grace Hospital.

1. Lundy, J. S.: *Clinical Anesthesia* (Philadelphia: W. B. Saunders Co., 1942) pp. 393, 583.
2. Isenberger, R.M.: The pharmacologic actions of intravenous procaine as an analgesic agent. Anesthesiology **10**:343-351, May 1949.
3. Hirschel, cited by Adams, R. C.: *Intravenous Anesthesia* (New York: Paul B. Hoeber, Inc., 1944), pp. 115-116.
4. Hatcher, R. A., and Eggleston, C.: The pharmacology of novocain. J. Pharmacol. & Exper. Therap. **8**: 385, July 1916.

from 60 to 200 drops a minute for a period of thirty to ninety minutes, until the patient obtains relief from pain, or adverse symptoms of toxicity appear. These toxic reactions depend on the total dose administered, speed of injection, and concentration of solution used. In true idiosyncrasy convulsions may occur and can be relieved by the administration of barbiturates. Pentothal sodium only in amounts sufficient to control the convulsions should be administered intravenously along with the administration of plenty of oxygen. Another form of reaction is respiratory difficulty of the asthmatic type, which is controlled by 2-4 minims of epinephrine (1:-1,000) given subcutaneously. Minor symptoms such as dizziness, slurred speech, apprehension, tremors, and mental confusion may be noted, as may lacrimation, a metallic taste, and slight lightheadedness.

Before administration of procaine the patient is always given a wheal test for sensitivity; the test is read in fifteen to twenty minutes. If further verification of lack of sensitivity is desired, a drop of the drug may be put in the patient's eye and the reaction observed.

Some workers prefer to use the 0.2 per cent concentration of procaine in normal saline or glucose solution in order to decrease the amount of fluid administered and the time of onset of action. When the 0.1 per cent solution is used, an optimal effect is generally seen within forty-five minutes and may continue for three to twelve hours after the end of the infusion, depending upon the severity of the pain. The total dose of procaine for relief of pain for twenty-four hours has been reported to be 0.5 to 10 Gm.

Investigations made by Richards[5] correlated vitamin C depletion, starvation, and improper electrolyte balance with procaine toxicity. To the elderly and poorly nourished patient who has lost fluids by dehydration and vomiting, liberal amounts of vitamin C and glucose are administered prior to the use of procaine, regardless of the technic by which it may be employed.

When procaine is used intravenously, it must be remembered that the margin between anesthesia and convulsions is narrow, and unless this margin can be widened, the use of procaine will be limited in the field of anesthesia. This can be accomplished by the addition of pentothal sodium, the dilution of procaine, and its use with *d*-tubocurarine chloride. This technic was developed by the independent work of Lundy, Gordon, and McLachlin.[6] Their first attempts failed because of the formation of a precipitate due to the concentration of the solution. Later it was found that pentothal sodium and procaine could be used together in more dilute solutions. With the use of this dilute pentothal sodium-procaine solution, no permanent ill effects have been observed. Preoperative barbiturate sedation also helps prevent reactions.

USE OF PROCAINE TO DIMINISH CARDIAC IRRITABILITY

The use of procaine intravenously during anesthesia to diminish cardiac irritability is of great importance to the anesthetist. Several investigators have shown that procaine applied locally to the heart reduces the irritability of the myocardium.

Factors contributing to cardiac irritability are: the psychic state of the patient, the anesthetic agent

5. Richards, K.: Abbott Bull., Aug. 1949.

6. Fraser, R. J., and Kraft, K. A.: Pentothal-procaine anesthesia. Anesth. & Analg. **27**: 282-286, Sept.-Oct. 1948.

used and technic employed, and the injection of certain drugs. Apprehension or emotional excitement, with the coincident outpouring of epinephrine into the circulation, is a definite factor contributing to hypersensitization of the cardiac conducting mechanism. When the anesthetic agent used is cyclopropane, chloroform, or ethyl chloride, the myocardium is particularly sensitized, and minute amounts of epinephrine may cause ventricular fibrillation. Reports of experiments indicate that the administration of procaine systemically diminishes this hyperirritability of the heart and can revert a shifted pacemaker to the sinus node.

In electrocardiographic studies performed during surgical anesthesia, all the common general anesthetic agents being used, Kurtz, Bennett, and Shapiro[7] found a high incidence of arrhythmia. In most of these cases the arrhythmias were not detected by the anesthetist at the peripheral pulse. This increase in cardiac irritability during anesthesia is such that certain drugs having definite action on the heart appear to cause toxic effects when doses considered therapeutic in the conscious individual are administered to the anesthetized patient.

In three of ten cases, reported by C. L. Burstein,[8] of patients with a pre-existing pathologic condition of the heart, who were operated on under procaine anesthesia, the following observations were made.

CASE 1.—The patient had constrictive pericarditis and was undergoing pericardectomy. The pulse was irregular. An electrocardiogram showed a transition from a sinus rhythm to a nodal rhythm. Procaine, 100 mg. in 1 per cent solution, was given intravenously. The pacemaker reverted to the sinus node forty seconds later. Eight minutes later ventricular fibrillation again occurred. Another 100 mg. procaine was given, and within one minute there was a return of normal pulse, which was maintained for the remainder of the operation.

CASE 2.—The patient was a woman, aged 43, undergoing thyroidectomy. The basal metabolic rate was plus 61. Her condition was complicated by signs of cardiac failure and pitting edema of both lower extremities. Before induction of anesthesia the blood pressure was 110 mg. Hg systolic and 70 mm. Hg diastolic; pulse was 100 beats a minute. Pentothal sodium, 0.5 Gm., was given for induction of anesthesia, followed by the administration of cyclopropane. Twenty-five minutes later the blood pressure was 200 mm. Hg systolic and 100 mm. Hg diastolic; pulse was 190 beats a minute and irregular. A dose of 100 mg. procaine in a 1 per cent solution was given intravenously. This was followed by a decrease in blood pressure to 130 mm. Hg systolic and 80 mm. Hg diastolic; pulse became regular at 86 beats a minute. Convalescence was uneventful.

CASE 3.—A man, aged 27, was to undergo inguinal hernioplasty. His physical status was good. The usual preanesthetic medication was given. Before anesthesia the blood pressure was 100 mm. Hg systolic and 70 mm. Hg diastolic; pulse rate was 60 beats a minute; respiratory rate was 20 a minute. Anesthesia was induced with cyclopropane. Eight minutes later the pulse rate decreased to 36; blood pressure and respiratory rate remained the same. The oxygen concentration was increased, and the pulse rate increased to 44 beats a minute. The administration of cyclopropane was discontinued, and ether was added to the anesthetic mixture. Five minutes later the pulse rate was 34; blood pressure was 68 mm. Hg systolic and 50 mm. Hg diastolic. Procaine, 100 mg. in 1 per cent solution, was given intravenously, and one minute later the pulse was 60 beats a minute; blood pressure was 110 mm. Hg systolic and 80 mm. Hg diastolic; respiratory rate ranged from 20 to 26 a minute. These levels were maintained for the remainder of the operation. Recovery was uneventful.

Various studies have revealed that, during general anesthesia with any of the commonly used anesthetic agents, a state prevails in which the central nervous system is de-

7. Kurtz, C. M.; Bennett, J. H., and Shapiro, H. H.: Electrocardiographic studies during surgical anesthesia. J. A. M. A. 106:434-440, Feb. 8, 1936.

8. Burstein, C. L.: The utility of intravenous procaine in the anesthetic management of cardiac disturbances. Anesthesiology 10:133-145, March 1949.

pressed but in which the heart becomes hypersensitive. Procaine tends to diminish this cardiac hyperirritability.

At Grace Hospital we have had a few experiences with the use of procaine intravenously. The following three cases are illustrative.

CASE 1.—The patient was to undergo gastric resection. Before the induction of anesthesia, it was noted that there was an irregularity in the pulse rate, which was 80-84 beats a minute. Forty-five minutes after the operation was started, the irregularity became pronounced. Atropine, gr. 1/150, was administered intravenously. The pulse was regular for about thirty minutes, but then became very irregular again. Procaine, 0.1 per cent solution, was started intravenously, 70 cc. being used. The pulse returned to the preoperative rate and remained at the same rate and quality for the remainder of the operation.

CASE 2.—During cesarean section with the patient under cyclopropane anesthesia, an irregularity in the pulse was noted. Procaine, 1 per cent solution, was administered intravenously very slowly. The pulse became regular and stable for the remainder of the surgical procedure.

CASE 3.—The patient was to undergo oophorectomy. The initial blood pressure was 140 mm. Hg systolic and 80 mm. Hg diastolic; the pulse rate was 74 beats a minute; the respiratory rate was 18. Anesthesia was induced with cyclopropane at 9:05 a. m. Intravenous administration of procaine, 0.1 per cent solution, was started at the same time. Intocostrin was also used. At 9:35 a. m. the pulse became rapid and irregular at 120 beats a minute; blood pressure was 210 mm. Hg systolic and 130 mm. Hg diastolic. Procaine was administered more rapidly, and the oxygen concentration was increased. Ten minutes later the pulse was 100 beats a minute; blood pressure was 160 mm. Hg systolic and 100 mm. Hg diastolic. Twenty minutes later an irregularity in the pulse was again felt. Cyclopropane and the intravenous administration of procaine were discontinued, and a mixture of nitrous oxide, oxygen, and ether was substituted. At the close of the operation at 10:10 a. m. the blood pressure was 110 mm. Hg systolic and 80 mm. Hg diastolic; respiratory rate was 24 a minute. It was the opinion of Dr. Myra Babcock that the cardiac irregularity had been the beginning of ventricular fibrillation and that it would have been controlled if a 1 per cent instead of a 0.1 per cent solution of procaine had been used; only 25 cc. of the 0.1 per cent solution was used. Recovery was uneventful.

The efficacious results from the use of procaine intravenously in overcoming cardiac irregularities manifested during general anesthesia have led to the belief that this therapy tends to minimize the state of cardiac hyperirritability produced by general anesthesia. This belief is substantiated by the beneficial results obtained by the administration of procaine intravenously as a prophylactic measure to diminish the incidence and severity of these cardiac arrhythmias.

PROCAINE IN ORTHOPEDIC SURGERY

Especially good results have followed the intravenous use of procaine in orthopedic surgery. Graubard and Peterson[9] reported its use in cases of fracture and dislocation with relief of pain before and after reduction, decrease in edema, and some clinical evidence of early healing. Analgesia was not sufficient for reduction of the fractures except in two cases. Post-traumatic edema diminished within forty-eight hours and frequently necessitated application of a smaller cast. Uniformly good results were obtained in sprains and varied results in traumatic arthritis.

DIFFERENTIAL DIAGNOSIS OF RUPTURED INTERVERTEBRAL DISK

The use of procaine intravenously also finds application in the differential diagnosis of ruptured intervertebral disk. No relief from pain is obtained when this condition exists. In six patients who received procaine intravenously for differen-

9. Graubard, D. J., and Peterson, M. C.: The therapeutic uses of intravenous procaine. Anesthesiology 10:175-187, March 1949.

tial diagnosis of ruptured intervertebral disk at Grace Hospital, we made the following observations:

In one patient there was *no* apparent relief from pain after the administration of procaine. Roentgenograms revealed a protruding intervertebral disk, and laminectomy confirmed the diagnosis.

In the next two cases *some* relief from pain was obtained. The first of these patients had improved mobility of the right arm. Roentgenograms showed a possible lesion of the intervertebral disk. No operation was performed. In the second patient roentgenograms revealed no gross pathologic condition. A myelogram revealed no evidence of block, and the patient was discharged.

Of the last three patients, the first was *relieved* by procaine injection, and spinal tap showed no evidence of block. The second patient had *definite relief* from pain, and roentgenograms showed no evidence of disease. The third patient had *relief* from pain, and a hemilaminectomy revealed no evidence of herniated disk; the final diagnosis was nerve root compression.

Although this series of cases is too small for any definite conclusions to be drawn from the results, it would tend to substantiate the findings that procaine administered intravenously can be useful in the differential diagnosis of ruptured intervertebral disk.

Other Uses of Procaine Intravenously

The use of procaine intravenously seems also to be particularly effective in the treatment of trauma. It blocks the primary neuron at the source of irritation, and the solution administered intravenously has proved to be seven to eight times more concentrated in traumatized tissue than in normal tissue. Peripheral pain usually causes reflex vasoconstriction in the area and also in other vascular areas. That vasospasm interferes with normal healing processes is well established; consequently, interference with the reflex arc after trauma will hasten recovery and rehabilitation of the injured patient.

Ezes[10] reported the administration of procaine to six women with eclampsia. In one of the patients a convulsion was followed by coma. Intravenous administration of 10 cc. of 1 per cent solution of procaine brought about a reduction of the blood pressure. There were no further convulsions, and the patient's vision improved after the second injection. All convulsions were fewer and less intense after injection of procaine. Ezes stated that this treatment is indicated particularly for primary eclampsia during labor and after delivery.

Procaine is also successfully used in the treatment of serum sickness. Dressler and Dwork[11] reported its use successfully in the treatment of penicillin reactions. Procaine has been used in place of morphine sulfate for postoperative pain, and a more comfortable convalescence follows. This drug is currently being used in many other types of diseases too numerous to mention.

Contraindications

Very little has been said about contraindications to this method of administering procaine, except in cases of sensitivity to the drug. Schrum[12] listed as probable contra-

(Continued on page 167)

10. Ezes, H.: Abbott Bull., Aug. 1949.
11. Dressler, S., and Dwork, R. E.: Reactions to penicillin. J. A. M. A. **133**:849, March 22, 1947.
12. Schrum, David: Intravenous procaine in children. Current Med. Digest, pp. 38-39, Aug. 1949.

PREOPERATIVE AND POSTOPERATIVE FLUID THERAPY

William J. Pendergrast, M.D.
Atlanta

As surgery becomes more radical, anesthesia and preoperative and postoperative care become more important. As Lord Moynihan said, "Surgery has been made safe for the patient, now let us make the patient safe for surgery." Rarely does an emergency occur that does not allow time for the correction of anemia or fluid imbalance. Fluid therapy falls into two major divisions: first, the correction of anemia and hypoproteinemia and, second, the maintenance of fluid and electrolyte balance. The patient receives fluids to prevent or overcome anemia, hypoproteinemia, or electrolyte imbalance before, during, and after operation. It cannot be too strongly emphasized that a patient who can take adequate fluid and food by mouth does not usually require parenteral administration of fluids. Too frequently fluids are routinely given parenterally when they are not needed and when they may even be harmful.

In preparing a patient for operation, the correction of anemia is of primary importance. The work of Clark et al[1] pointed out that the chronically ill or debilitated patient usually has a deficiency of hemoglobin as well as a deficiency of tissue and plasma proteins. In studies on chronic shock they showed that in such a patient a decreased blood volume will mask anemia and hypoproteinemia. This state of masked anemia or chronic shock makes the patient extremely susceptible to shock and all the attending complications. They suggested that, in preparing the debilitated patient for operation, it is not safe to depend solely on the erythrocyte count, hemoglobin, and hematocrit reading in making a decision as to whether transfusion will be needed preoperatively. They stated that it is necessary to determine the total blood volume of the patient, estimate what his blood volume would be if his weight were normal, and then give transfusions to increase his blood volume to that level. They found that for a patient with fairly severe malnutrition an average of 2,700 cc. blood was necessary to increase the blood volume to the estimated normal. This increase in blood volume was attended by definite but asymptomatic hemoconcentration. Patients so prepared withstand prolonged and extensive surgical procedures with remarkably little change in blood pressure, pulse, and respiration and with few postoperative complications.

The value of correcting anemia by transfusion was also emphasized

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Read before the Southeastern Assembly of Nurse Anesthetists, Biloxi, April 27, 1949.

1. Clark, J. H., and others: Chronic shock: Problem of reduced blood volume in chronically ill patients. Ann. Surg. 125:618-646, May 1947.

by Whipple and Madden.[2] They found that plasma and tissue proteins are replaced very slowly until an existing anemia is corrected. When the hemoglobin becomes normal, plasma proteins are replaced rapidly. Since there is a definite ceiling to the rate at which new hemoglobin is formed, it appears that much time can be saved in preparing a debilitated patient for operation by giving transfusions sufficient to create a normal blood volume and then feeding large amounts of proteins to overcome plasma and tissue deficiencies. Protein given by mouth is by far the most economical and satisfactory method of administration. Elman[3] showed that 375 Gm. protein daily for ten days is required to increase the serum protein from a level of 5 mg. per 100 cc. to the normal level of 7 mg. per 100 cc. Thirty grams of protein is deposited in the tissues for every gram that goes into the serum. This eliminates plasma as a source of protein for economic reasons.

Amino acid preparations are quite satisfactory for use in maintaining nitrogen balance, but the administration of 300-400 Gm. protein daily by the intravenous method is also impracticable. To avoid postoperative electrolyte imbalance a serum protein level of 6.5 or 7 mg. per 100 cc. should be attained. Clinically, edema does not appear until the level of serum protein is below 5.5 mg. per 100 cc.; however, there is incipient edema when the level is below 6.5 mg. per 100 cc. Edema developing at the site of operative trauma may cause delayed healing, wound disruption, evisceration, or herniation. Such edema may obstruct a mechanically perfect intestinal anastomosis.

Large quantities of proteins must be administered orally to overcome a severe protein deficiency. Many preparations are available to supplement the diet. Since most of these are made from milk, ordinary skimmed milk powder is an inexpensive and satisfactory source of proteins and carbohydrates. Forced feeding can be accomplished by the administration of proteins and carbohydrates through a Levine tube. In this manner a daily intake of 300-400 Gm. protein may be insured.

When we consider the problem of fluid and electrolyte balance before operation, let us assume that the daily basal fluid requirement is about 2,700 cc. Of this amount, 1,400 cc. urine with a specific gravity of 1.010 is needed to excrete the average daily waste of 35 Gm. About 1,000-1,300 cc. of additional fluid is lost in perspiration, respiration, and defecation. If fever, hyperthyroidism, or a hot humid atmosphere is present, far larger quantities are lost. A temperature of 101 F. may cause a patient to lose an additional 1,000 cc. daily through perspiration. Perspiration varies with the season, the temperature of the operating room, and the extent of draping and covering of the patient. A person lying quietly in bed in a hot humid hospital may perspire as much as 4,000 cc. daily.

In preparing the dehydrated patient for operation, it is first necessary to determine the type of dehydration and the type and quantity of fluid required to return the fluid balance to normal. In simple dehydration—that is, when the patient is unable to take fluids, possibly be-

2. Madden, S. C., and Whipple, G. H.: Plasma proteins: their source, production and utilization. Physiol. Rev. 20:194-217, April 1940.

3. Elman, Robert, and Weiner, D. O.: Intravenous alimentation, with special reference to protein (amino acid) metabolism. J.A.M.A. 112:796-802, March 4, 1939;; Elman, Robert; Weiner, D. O., and Bradley, E.: Intravenous injections of amino acids (hydrolyzed casein) in postoperative patients. Ann. Surg. 115:1160-1165, June 1942.

cause of esophageal obstruction—one finds severe thirst, decreased urine output, and dry mucous membranes. Here the need is largely for distilled water with glucose. Normal saline solution will not relieve the thirst, which is due to simple loss of water. This is true because the kidneys are capable of conserving salt when the intake is insufficient. The dehydrated patient will excrete only traces of chloride in the urine. Therefore, the administration of 5 Gm. salt daily is more than adequate for normal needs. Since it is impossible to be sure of the amount of salt lost through perspiration, it is suggested that, on warm days or when the patient has a fever, 10 Gm. salt, that is, 1,000 cc. of normal saline solution, be given daily. If this is more salt than is needed, the kidneys will excrete the excess. After the administration of saline, glucose in distilled water should be given until thirst is overcome and urinary output reaches at least 1,000 cc. daily.

Often dehydration is complicated by a deficiency of electrolytes. When the patient vomits, the effect on the body is loss of water, chlorides, and small amounts of sodium. The practice of allowing a patient undergoing gastric suction to drink water freely causes the washing out of large amounts of chlorides. In acute intestinal obstruction, considerable fluid and electrolyte loss into the distended paralytic bowel occurs. From a high intestinal, biliary, or pancreatic fistula and from severe diarrhea, there is loss of fluids containing larger amounts of alkalies.

Electrolyte imbalance from excessive loss of chlorides, such as occurs in vomiting with pyloric obstruction, should be corrected by the administration of saline or Ringer's solution. A high intestinal or biliary fistula or severe diarrhea tends to cause acidosis from loss of sodium. This is best treated by the use of Hartman's solution of 1/6 molar sodium lactate. Studies made at Grady Hospital in Atlanta[4] showed that severe acidosis can be quickly and safely overcome by the addition of two teaspoonfuls of Arm & Hammer baking soda to 1,000 cc. of 5 per cent glucose. This is locally known as the "Arm & Hammer" treatment and produces dramatic improvement in the acidosis of uremia or renal shutdown from prolonged shock or transfusion reaction.

For years an attempt was made to calculate by laboratory tests (urine specific gravity, plasma chlorides, carbon dioxide-combining power, hematocrit, etc.) the exact type and quantity of fluids needed by the dehydrated patient. For clinical application, a rule based on variations in the plasma chlorides was offered by Coller and Maddock.[5,6] This rule is now recognized by them as being highly inaccurate and dangerous. Chlorides were found to vary greatly in different pathologic states. Because of these variations, excesses of saline were occasionally given. In debilitated patients and postoperative patients this led to salt and fluid retention and attending complications. Coller and co-workers[6] subsequently showed that the various laboratory tests can only indicate the type of electrolytes needed. They pointed out that the amount required depends more on the patient's clinical response than on any laboratory test. For instance, a man has obstruction and vomiting of two weeks' dura-

4. Personal communication to the author.
5. Coller, F. A., and others: Replacement of sodium chloride in surgical patients. Ann. Surg. 108:769-782, Oct. 1938; Coller, F. A., and Maddock, W. G.: Study of dehydration in humans. Ann. Surg. 102:947-960, Nov. 1935.
6. Coller, F. A., and others: Postoperative salt intolerance. Ann. Surg. 119:533-542, April 1944.

tion. He has lost weight and is thirsty; his temperature is 97 F.; the pulse is very soft and thready; blood pressure is low; and he is stuporous. The specific gravity of the urine is high, 1.028; plasma chlorides are low, 400 mg. per 100 cc.; carbon dioxide-combining power is elevated, 90 volumes per cent. This man is immediately given 1,000 cc. saline solution and 2,000 cc. 5 or 10 per cent glucose solution to overcome thirst and increase urine output. The administration of glucose in distilled water is repeated until thirst is alleviated. The administration of saline is repeated, but as soon as his pulse becomes stronger, his temperature rises, and his mind clears, the administration of saline is stopped, no matter what the blood chloride is. On subsequent days sufficient glucose in distilled water is given to allay thirst and increase urine output to 1,000 cc. Five grams of salt as saline or Ringer's solution is given, plus saline as replacement for loss by suction or excessive perspiration. If his clinical condition is good, no attempt is made to change a low chloride level.

During any surgical procedure lasting for an hour or longer, the administration of 1,000 cc. glucose in distilled water is always of value. This is true because the patient will have a period of about twenty-four hours during which very little fluid will be taken by mouth. Here again it is well to emphasize that the average patient undergoing the average surgical procedure will be able to take fluids by mouth after the first twenty-four hours and should not require fluids given parenterally.

During an operation, if excessive bleeding is anticipated, blood should be ready for administration in the operating room. The treatment of shock should first be the prevention of shock. A patient returning from the operating room with a systolic blood pressure of 90 mm. Hg or below should receive transfusions until the pressure reaches a satisfactory level. The benefits of saline solution are transient. Traumatic shock is best treated by the administration of plasma. When the systolic blood pressure is below 80 mm. Hg, it is considered insufficient for proper oxygenation. When the level decreases to below 60 mm. Hg, the condition is critical. Studies at Emory University Hospital[7] demonstrated the life-saving value of intra-arterial blood transfusion. When shock and blood loss are sudden, severe, and seemingly irreversible, this measure will quickly force blood into the circulation and bring a patient in a critical state of shock back to a fairly stable condition.

No matter how carefully a patient is prepared preoperatively, protein deficiency and electrolyte imbalance must be watched for postoperatively. Protein deficiency develops in a surgical patient because there is a decreased intake; there is loss of proteins through internal exudates or from wound orifices or fistulas; and after severe trauma and prolonged anesthesia of any type (notably ether and cyclopropane), there may be albuminuria lasting for as long as three or four days.[8] Studies on the average surgical patient show protein decreases of 1 mg. per 100 cc. during the first week, in spite of careful replacement of total blood lost at operation. If the patient is well prepared preoperatively, this loss can

7. Robertson, R. L.; Trincher, I. H., and Dennis, E. W.: Intra-arterial transfusions; experimental and clinical considerations. Surg. Gynec. & Obst. 87:695-704, Dec. 1948.

8. Coller, F. A., and others: Effects of ether and cyclopropane anesthesia upon renal function in man. Ann. Surg. 118:717-727, Oct. 1943.

easily be offset by the parenteral use of amino acid preparations.

During the early postoperative period one must not upset electrolyte balance. After a prolonged operation and anesthesia there is a renal shutdown lasting from twenty-four to forty-eight hours. During this period in many patients, especially elderly patients or those who are poor risks, a condition develops that has been called postoperative salt intolerance. This condition was first described by Troute[9] and Evans[10] and later gained wide publicity through the work of Coller and coworkers.[6] They pointed out that normal kidneys are capable of excreting as much as 50 Gm. salt daily. During the period of postoperative urinary suppression, this ability is lost. The administration of excessive amounts of saline during this early postoperative period may give rise to the symptom complex of salt intolerance. The patient has weakness, disorientation, anorexia, nausea, vomiting, distention, deep respiration, and decreased urinary output. The nonprotein nitrogen level rises, and the carbon dioxide-combining power falls. The condition progresses to acidosis, and unless saline administration is stopped, the patient will die.

Because of salt intolerance, the water requirements in the first forty-eight hours postoperatively should be met entirely with glucose in distilled water. Saline solution should be given only to replace losses from suction or fistulous drainage. Because this drainage contains about 5 Gm. salt in 1,000 cc., volume for volume replacement should be made with 0.5 per cent normal saline solution instead of the usual physiologic, or normal, saline solution. The widespread tendency to give normal saline solution to all patients requiring parenteral administration of fluids, with no regard for the actual salt lost, causes other complications: local and generalized edema, delayed healing with increase in number of eviscerations and/or incisional herniations, decreased motility of the gastrointestinal tract, and obstruction of an intestinal anastomosis by edema.

9. Troute, H. H.: Proctoclysis; an experimental study. Surg. Gynec. & Obst. 16:560-562, May 1913.
10. Evans, G. H.: The abuse of normal salt solution. J.A.M.A. 57:2126, Dec. 30, 1911.
6. Coller, F. A.: loc. cit.

SUMMARY

It should be emphasized that blood, proteins, carbohydrates, and vitamins should be used in large quantities in preparing debilitated patients for operation and in caring for them postoperatively. When the blood constituents are normal, electrolyte balance is less difficult to maintain. The management of dehydration and electrolyte imbalance in the preoperative and the postoperative period may be summarized in this manner:

1. Chart and record fluid intake and output.
2. Check specific gravity and urinary chlorides.
3. Maintain urinary output at 1,000-1,500 cc. daily.
4. Avoid giving excessive amounts of salt.
5. Give no saline solution in the first twenty-four to forty-eight hours postoperatively except to replace abnormal loss.
6. Check blood chemistry every forty-eight hours until values approach normal.
7. Do not depend entirely on the laboratory.

There is no substitute for clinical observation. It has been said with good reason that "the surgeon of today should be primarily a physiologist and secondarily a technician."

POSTOPERATIVE URINARY SUPPRESSION DUE TO ANOXIA

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Detroit

Some of the effects of anesthesia on renal function have been recognized for as long as fifty years. Most of these effects are temporary and have been summarized by a host of writers. Experienced anesthetists are no doubt fully cognizant of these phenomena, and a recapitulation is not within the scope of this presentation. Instead, we shall attempt to describe an unpredictable type of postoperative renal failure to which attention was directed during World War II. Since the cessation of hostilities, intensive studies have resulted in a much more comprehensive understanding of this condition. Similar, if not identical, forms of renal insufficiency continue to plague clinicians in almost every medical specialty, and their importance cannot be overemphasized.

During the London "blitz," British surgeons discovered that many patients died from uremia several days after crushing injuries to muscles of the extremities. Autopsies on these individuals revealed distinctive lesions in otherwise normal kidneys. The same course of events had been observed during World War I but had been forgotten or ignored until the English reports stimulated renewed interest. Soon this peculiar symptom complex became known as the "crush syndrome."

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Mallory,[1] of the U. S. Army, found the same renal changes in 10 per cent of the first thousand autopsies reviewed by him in the Mediterranean Theater. Angevine and Harmon[2] discovered identical lesions in 15.2 per cent of 1,065 battle casualties.

While serving as a surgical consultant to the U. S. Army, E. D. Churchill[3] summarized the experiences of many observers when he wrote, "By excellent forward surgery and the liberal use of whole blood transfusions as well as plasma, we are saving lives, but also keeping certain men alive temporarily only to display later kidney damage. There has been either complete anuria with death or . . . a fall in urinary output . . . with ultimate recovery of kidney function. This phenomenon is not unique to the 'crush syndrome' but may occur in any wounded man who experiences a long period of greatly reduced volume flow."

Finally, in 1946, Lucké[4] crystallized most of the clinical and pathologic data after reviewing 538 fatal cases of this type at the Army Institute of Pathology in Washington. It was obvious that the renal changes were not specific for the

1. Mallory, T. B.: Hemoglobinuric nephrosis in traumatic shock. Am. J. Clin. Path. 17: 427, 1947.
2. Angevine and Harmon, cited by Lucké.[4]
3. Churchill, cited by Van Slyke.[6]
4. Lucké, B.: Lower nephron nephrosis. Mil. Surgeon **99**: 371, 1946.

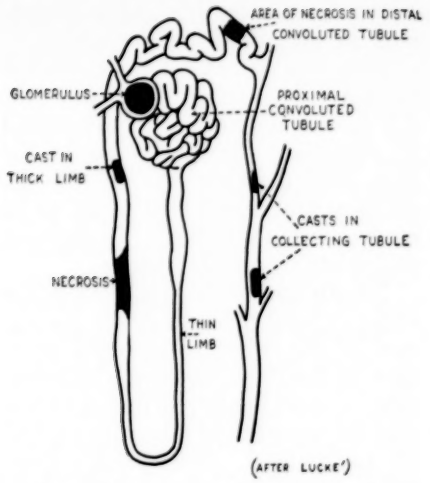

Fig 1.—Diagrammatic summary of typical histologic findings in lower nephron nephrosis. Areas of necrosis occur in the distal tubules, and heme casts are found in the lower nephrons and collecting tubules.

"crush syndrome." Identical lesions were found after intravascular hemolytic reactions, crushing injuries, burns, nontraumatic muscular ischemia, sulfonamide intoxication, toxemia of pregnancy, heat stroke, blackwater fever, and some types of poisoning. The initiating factor seemed to be destruction of either tissue or blood.

The common clinical manifestations were shock, vomiting, and the excretion of little or no urine. With the decrease in urinary output, the patient had bloody or smoky urine containing albumin and casts, increased nonprotein nitrogen, decreased alkali reserve, and not infrequently hypertension and edema.

The kidneys in such cases were swollen and increased in weight, and the cortex appeared unusually pale. Microscopically, there was little or no disturbance of the glomeruli or proximal tubules. The typical histologic findings are summarized diagrammatically in figure 1. Degeneration and necrosis occurred in the distal tubules, while casts of blood pigment were found in the collecting tubules and in the lower nephrons. Lucké, therefore, introduced the term "lower nephron nephrosis" because he thought it was most descriptive of this condition.

In general, Lucké's terminology has enjoyed widespread popularity, but other writers have preferred hemoglobinuric nephrosis, hepatorenal syndrome, prerenal azotemia, anoxic nephrosis, or even "shock kidney." It is obvious, therefore, that there has been much investigation and even more speculation regarding the pathogenesis of this type of renal failure.

Corcoran and Page[5] showed that there is an irregular flow of blood in the kidney with any significant fall in blood pressure. They stressed the importance of renal ischemia in producing the tubular damage that is typical of this syndrome.

Van Slyke[6] claimed that identical renal changes may develop after any type of shock, whether it be caused by hemorrhage, trauma, or dehydration. He identified two important stages in the effect of shock on the kidney: (1) ischemia and (2) renal damage. There is no sharp division between these two phases. When the systemic blood pressure falls, the renal blood flow is diminished with an attendant decrease in renal function. Complete anuria may develop, but if the renal ischemia is not too great or prolonged, restoration of normal circulation will be followed by a return of normal kidney function. According to Van Slyke, a decrease in systolic blood pressure of 40 to 60 mm. Hg is "sufficient to cause anuria be-

5. Corcoran, A. C., and Page, I. H.: Crush syndrome: Post-traumatic anuria. J. A. M. A. **134**: 436, 1947.

6. Van Slyke, D. A.: Effects of shock on the kidney. Ann. Int. Med. **28**: 701, 1948.

cause the pressure is insufficient to maintain glomerular filtration."

If the low blood pressure exists long enough, restoration of the circulation of the kidney will *not* be followed by normal renal function because of permanent damage to the tubules. This is thought to be due to anoxia of the cells. As Haldane once said, "Anoxia not only stops the machinery but it may wreck the machine." Complete or partial renal failure, due to irreversible cellular changes, may last until the patient dies from uremia. If the damage is less extensive, there may be some gradual return of renal function, and the patient may recover even though his renal reserve may be depleted.

Still other writers emphasize the possible role of liberated toxins that act directly on the renal tissues and produce necrosis. Regardless of the precise theory of choice, the consensus is that the ultimate result of the inadequate circulation, or any other destructive process, is anoxia of the tubular epithelium.

Since the classic lesion in lower nephron nephrosis is in the distal tubules, persistent renal failure is thought to be due to the inability of the kidneys to concentrate the glomerular filtrate. Under normal circumstances this fluid eventually is voided, but owing to the damaged tubules, it leaks through the walls, and oliguria or anuria results.

With this background, we hope that everyone can appreciate the fact that any precipitous decrease in blood pressure is capable of producing the lower nephron syndrome with its varying degrees of urinary suppression, depending solely on the severity of the renal anoxia. Since hypotension may develop in any patient during almost any type of surgical procedure, it is this type of patient in whom we hope to interest you as anesthetists and as guardians of human blood pressures.

This type of renal complication is not a development of the atomic age. All of us, undoubtedly, have failed to recognize the syndrome in the past, and it behooves all of us, surgeon and anesthetist alike, to be more alert regarding its ominous potentialities in the future.

No one can be dogmatic about the exact critical point in either the extent of the decrease in blood pressure or the duration of the hypotension. Consequently, no one can forecast the ultimate postoperative effect on any patient. It may vary from mild transient oliguria to fatal anuria, depending on the degree of permanent renal damage.

Prevention of Lower Nephron Syndrome

As might be expected, our primary concern is the prevention of the lower nephron syndrome. Each of you as an anesthetist may have a most important role in prophylaxis. Prompt recognition of decreasing blood pressure during or after any surgical procedure is imperative. Any period of shock must be shortened as much as is humanly possible. Even though the anesthetist may be unable to control all of the individual factors that lead to a decrease in blood pressure during an operation, he occupies the key position for early recognition of unstable pressures.

Blood volume must be restored without delay, and there is no satisfactory substitute for the administration of whole blood to the patient who is bleeding. If it is your prerogative to start intravenous fluid administration, including whole blood transfusion, by all means do so promptly before any profound degree of shock can develop.

Too frequently there is blind faith in the liberal administration of pressor substances. While adrenalin, neosynephrin, arterinol, and allied drugs are invaluable additions to the armamentarium of the anesthetist, one must not forget that they cannot replace whole blood. Such agents should be used only as temporary measures while preparations are being made to give transfusions to those patients who may be victims of excessive bleeding.

With the increasing popularity of the continuous drip method for administering solutions containing vasopressor drugs, hemorrhage may be masked by a reasonable blood pressure. Constant evaluation of the entire picture is mandatory.

One word of caution, however, to those who administer whole blood. Administration of incompatible blood also is capable of producing the lower nephron syndrome. One cannot overstress the importance of making sure that every patient is given the blood that he is scheduled to receive. Few mistakes can be more tragic than the administration of the wrong blood in panic to a patient in a critical condition.

Overzealous intravenous administration of all types of fluids likewise must be condemned. Patients have been "drowned" in pulmonary edema merely because excessive loads of fluids have been poured into their precarious circulatory systems.

Needless to say, *every* decrease in blood pressure during an operation is not necessarily due to hemorrhage and surgical trauma. Hypotension resulting from the anesthesia per se, e.g., high spinal, can be equally disastrous and must be treated without delay.

If despite all alertness and the best efforts postoperative urinary suppression occurs, there are several cardinal points in appropriate therapy. These hardly concern anesthetists, but we mention them briefly to complete this clinical picture.

In the past anuric patients usually were given prodigious amounts of fluids in the hope of initiating diuresis. As a result, most of them died from pulmonary edema. Now it is customary to replace only the amount of fluid lost each day through normal channels. If there is no vomiting, diarrhea, or profuse sweating, the insensible loss seldom exceeds 1,200 cc. per day.

Every effort is made to maintain satisfactory electrolyte balance. Special care is taken to avoid acidosis. Patients are digitalized at the first indication of impending cardiac decompensation.

Little attention is paid to an elevation of nonprotein nitrogen unless it continues to increase after ten days. Many renal lesions subside and the kidneys again start to function after five to ten days. Those that persist beyond that time are treated by a variety of measures designed to prolong life in the hope of permitting delayed return of renal function. These include renal decapsulation, peritoneal lavage, gastric and duodenal lavage, and the well publicized artificial kidney. Each of these methods has its champions, but none of them is perfect. Detailed consideration of their relative merits and limitations would do little more than add confusion to this discussion. We much prefer to emphasize further the importance of prophylaxis by citing a few illustrative cases.

CASE 1.—A man, aged 45, had a total pancreatoduodenectomy for carcinoma (fig. 2). The operation was performed under fractional spinal anesthesia and required eight hours, during which time the blood pressure was imperceptible for a total of fifty minutes, despite the fact that the patient received 8 pints of

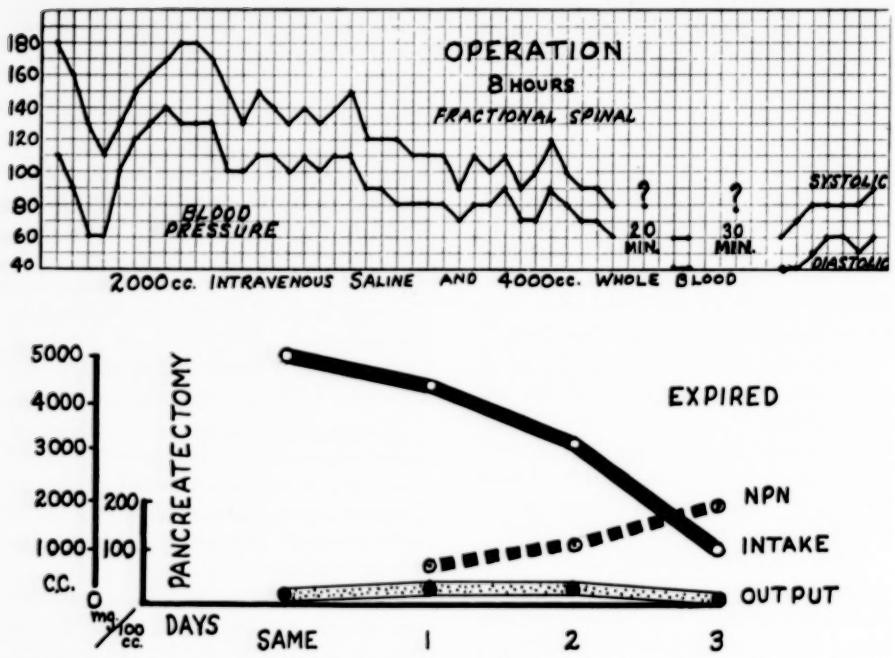

Fig. 2.—Lower nephron syndrome confirmed by autopsy three days after a long and shocking total pancreatectomy for carcinoma in a man, aged 45.

whole blood and 2,000 cc. of saline solution. The fluid intake postoperatively was more than adequate, but he excreted only a few ounces of urine. The nonprotein nitrogen increased rapidly, and he died early on the third day after operation. Autopsy showed a typical lower nephron syndrome.

Case 2.—A man, aged 52, with hypertension had a thoracolumbar sympathectomy under pentothal sodium anesthesia (fig. 3). The operation required more than three hours, during which time the blood pressure was extremely labile. For twenty minutes it was unobtainable. After the patient was returned to his room, he had further severe hypotension. No urine was excreted during the first twenty-four hours and only a minute amount during the following day. The nonprotein nitrogen was increased above normal limits, but the patient gradually put out more and more urine until his fluid balance and blood chemistry were normal on the seventh postoperative day.

Case 3.—A man, aged 41, with pulmonary tuberculosis was subjected to a thoracoplasty (fig. 4). The operation was completed with reasonable dispatch under ethylene and cyclopropane anesthesia. There was some decrease in blood pressure, but it seemed to remain stabilized at 120 mm. Hg systolic and 70 mm. Hg diastolic. One pint of blood was given during the procedure. After operation he excreted only a few cubic centimeters of urine. The fluid intake was kept within the range of what seemed to be necessary to replace the insensible loss, until the eighth day when gastric lavage was instituted. This reduced the nonprotein nitrogen reading from 195 to 165 in twenty-four hours, but the profound urinary suppression was unaltered. He died on the tenth postoperative day. Autopsy confirmed the diagnosis of lower nephron nephrosis and revealed terminal pulmonary edema. In all probability the blood transfusion initiated the renal damage, although the fluctuation in blood pressure may have been more significant than one would suspect.

Case 4.—A woman, aged 47, had a laminectomy and spinal fusion that required two hours and thirty-five minutes (fig. 5). She received 2,000 cc. of whole blood during the operation, and the fact that jaundice developed postoperatively suggested that her temporary renal insufficiency was due to intravascular hemolysis rather than to any mild degree of

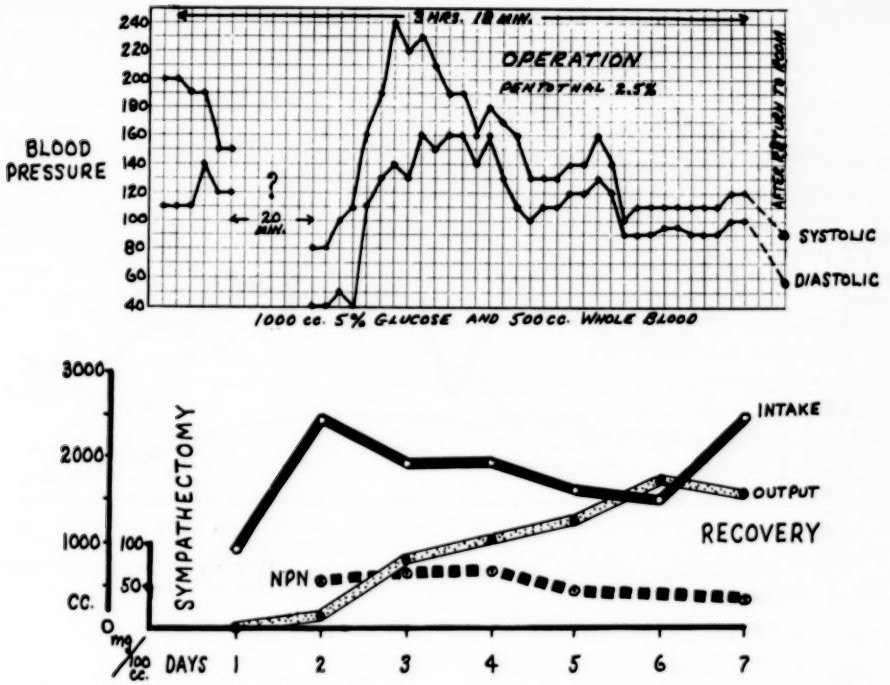

Fig. 3.—Temporary renal insufficiency in a man, aged 52, after sympathectomy, during which there was marked fluctuation in blood pressure.

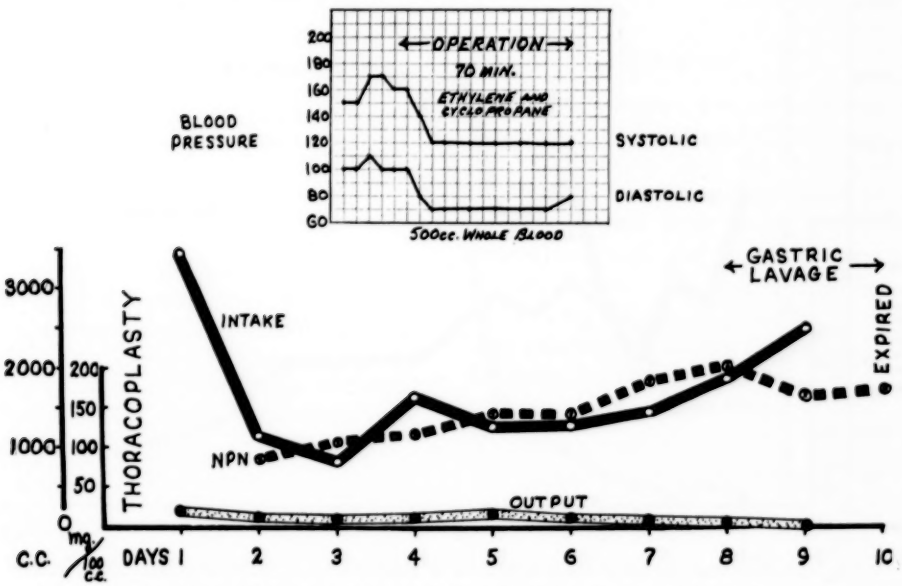

Fig. 4.—Lower nephron syndrome in a man, aged 41, after thoracoplasty. Autopsy showed terminal pulmonary edema.

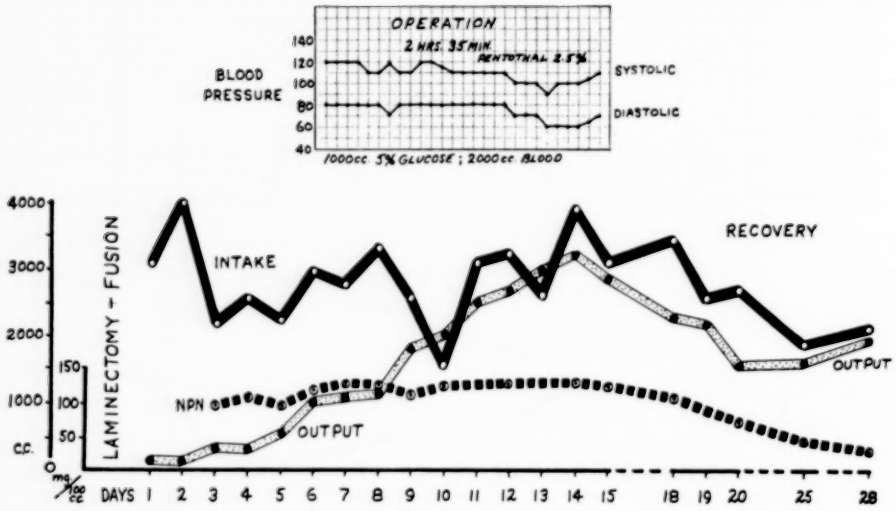

Fig. 5.—Temporary oliguria in a woman, aged 47, following laminectomy and spinal fusion during which 2,000 cc. of whole blood was administered.

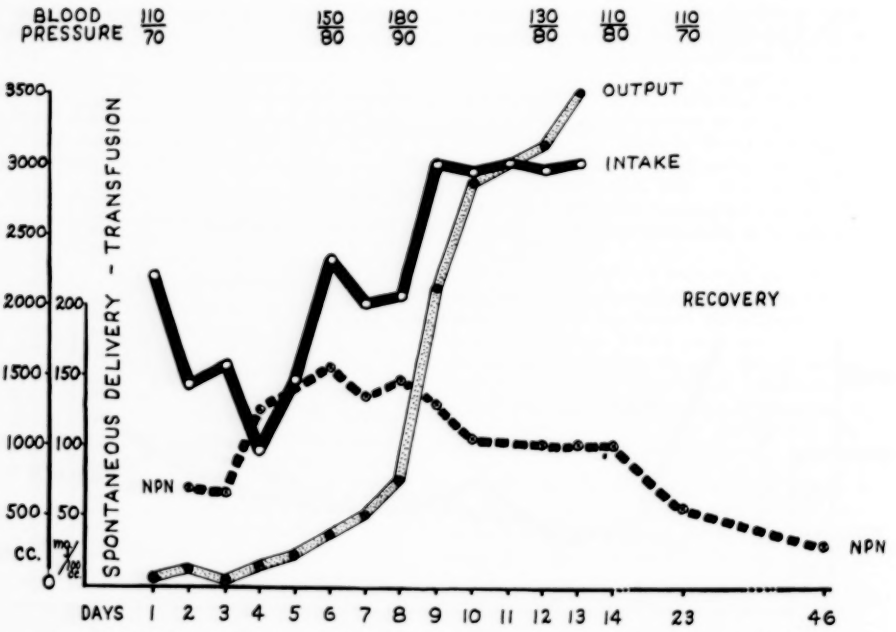

Fig. 6.—Nonfatal urinary suppression after delivery of a multipara, aged 35. Lower nephron syndrome was due to administration of incompatible blood.

shock. As noted on the graph, there was a gradual increase in urinary output, and the nonprotein nitrogen decreased until fluid balance and blood chemistry were normal on the twenty-eighth postoperative day. This case illustrates urinary suppression after comparatively inconspicuous changes in blood pressure.

CASE 5.—A multipara, aged 35, had spontaneous delivery of a stillborn child at six months. Because of blood loss she was to receive 500 cc. whole blood after being returned to her room. After she had received 100 cc., it was discovered that she was being given the wrong blood. Although the transfusion was stopped immediately, almost complete anuria developed (fig. 6). The nonprotein nitrogen steadily increased until it reached 155 on the sixth day. Blood pressure, which had previously been normal, increased to 180 mm. Hg systolic and 90 mm. Hg diastolic. Diuresis finally started, and from the tenth to the thirteenth day she passed amazing quantities of urine. However, the nonprotein nitrogen did not become normal until the forty-sixth day. Fortunately, a tragedy was averted in this instance, but let it be a lesson to anyone and everyone who finds it necessary to administer whole blood at any time. Other patients have been less fortunate.

It is apparent that the anesthetist is inheriting many responsibilities that concern the destiny of surgical patients. Prophylaxis against certain types of the lower nephron syndrome is definitely in the hands of the person at the head of the operating table.

SUMMARY

Renewed interest in postoperative renal failure has lead to enlightening studies indicating that anything capable of causing renal anoxia may result in the untimely death of the patient from uremia.

Included among the many etiologic factors is a severe or prolonged decrease in blood pressure, such as may occur in any operating room.

Early recognition of incipient hypotension and prompt institution of suitable corrective measures are vital.

Special warning is given to everyone that there is no substitute for whole blood in combating shock due to hemorrhage. Vasopressor drugs must be used only as temporary measures, and their use must not be allowed to mask the systemic effects of blood loss.

The injudicious intravenous use of fluids may cause fatal pulmonary edema, and the administration of incompatible whole blood may produce the very lower nephron nephrosis that we are trying to avoid.

PROCAINE

(Continued from page 154)

indications the presence of liver damage, myasthenia gravis, epilepsy or a history of epilepsy, and recent head injury.

SUMMARY

The intravenous use of procaine has a wide and varied field of clinical application, but the primary concern of the nurse anesthetist in relation to this drug is its use in connection with and as an adjunct to anesthesia. The other factors concerning the use of procaine have been mentioned because the anesthetist may be called upon to administer it to patients not under general anesthesia.

It must be remembered that, in spite of the amazing results obtained from the intravenous use of procaine, it is not a cure all and has its limitations.

OXYGEN LACK, CARBON DIOXIDE EXCESS, AND ASPHYXIA

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You, as anesthetists, and I, as a physiologist, have an important interest in common—we would like to understand the process of pulmonary respiration. I am interested out of general curiosity about how the complex human organism operates; you are interested because interference with the process necessitated by anesthesia should be minimal, and you are frequently called upon to maintain the process when Nature's own control breaks down. If you have to pinch-hit for Nature, it is a great consolation to know what Nature's objectives are.

We may approach our problem by asking three fundamental questions and discussing their answers. The three questions are: (1) What are the functions or purposes of respiration? (2) In what ways may respiration be disturbed, and how may these be detected? (3) What are the consequences of inadequate respiration?

Let us examine the purposes of respiration first from the limited standpoint of the lungs themselves; later we will consider them from the standpoint of their use to the whole organism.

Functions of Respiration

The specific function of the respiratory system is to accomplish the exchange of the respiratory gases, oxygen and carbon dioxide, between the external atmosphere and the circulating blood. Two separate but successive processes are involved in this exchange; the first is called *pulmonary ventilation,* and the second may be called *alveolar transfer.*

Pulmonary ventilation consists of maintaining an alternating flow of air between the surrounding atmosphere and the tiny lung sacs known as alveoli. It depends upon the mechanical bellows-like action of the lungs and thorax and associated muscles. Pulmonary ventilation is measured in terms of liters of air inspired (or expired) a minute. It is the total volume of air displaced every minute, regardless of the depth of individual breaths or their frequency. It averages at rest about 7 L. a minute, but it may be reduced by sleep to 5-6 L. a minute and may be increased by violent exercise to as much as 120 L. a minute.

Alveolar transfer, on the other hand, consists of the diffusion of oxygen and carbon dioxide between the air in the alveoli and the capillary blood, across the alveolar wall. It depends not only on diffusion and permeability but also on the capillary blood flow in the lungs. Alveolar transfer may be measured in terms of cubic centimeters of oxygen consumed a minute, or cubic centimeters of carbon dioxide eliminated a minute, since both diffuse

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across the alveolar wall. It averages at rest about 275 cc. a minute for oxygen consumption, somewhat less during sleep and anesthesia, and as much as 3,300 cc. a minute during violent exercise.

The job of the lungs, therefore, is to transfer respiratory gases. The *rate* at which this process is carried on is determined not locally by the lungs themselves but by the requirements of the body as a whole. These requirements vary widely as we have already seen in the case of physical exercise. But the requirements also vary with the body's temperature, endocrine status, acid-base balance, accessibility to oxygen, etc. Pulmonary ventilation, therefore, undergoes marked adjustments in accordance with these various demands.

The diverse pulmonary adjustments may be divided into two general types depending upon their purposes. The first type is called *metabolic,* because its purpose is to satisfy demands of the body for changes in its rate of metabolism. Thus in exercise, when metabolism is increased, the demand for extra oxygen uptake and carbon dioxide elimination results in an increase in both ventilation and alveolar transfer. The same is true in hyperthyroidism. It is only to be expected that in these conditions the ventilation and oxygen consumption should change proportionately so that the ratio of the two remains unaltered. This ratio has a particular name. It is called the *ventilation equivalent for oxygen.* Specifically, it is the liters of air breathed for each 100 cc. of oxygen absorbed. At rest, during sleep, in hyperthyroidism, and during moderate exercise, it remains constant at about 2.5 L. of air breathed for each 100 cc. of oxygen absorbed. This constant ventilation equivalent for oxygen is the identifying characteristic of metabolic adjustments of respiration.

The second type of pulmonary adjustment to body needs is called *compensatory,* because its purpose is to help the body compensate for some disturbance in either its internal or its external environment. If a healthy person is exposed to an altitude where the oxygen in the air is decreased, pulmonary ventilation is slightly augmented. The effect is to improve the oxygen supply to the lung alveoli and thus to compensate in part for the deficiency of the environmental air. This occurs even though the person is at rest without any change in rate of metabolism or oxygen consumption. Obviously, this increase in ventilation without a corresponding increase in metabolism means that the ventilation equivalent for oxygen must increase. Similarly, if the inspired air is altered by including carbon dioxide, the pulmonary ventilation is markedly increased. The effect again is to compensate in part for the defect in the inspired air by preventing an equal increase in the carbon dioxide in the lung air. This response also occurs even if the person is at rest, so that the ventilation equivalent for oxygen increases. Finally, a patient with diabetic acidosis suffers from a disturbance in his internal environment consisting of an increase in the hydrogen ion concentration of the blood. This likewise provokes an increase in ventilation, which serves to eliminate the volatile carbonic acid from the blood and thus to correct in part the abnormal hydrogen ion concentration of the blood. Again this happens even at rest with constant oxygen consumption, and the ventilation equivalent for oxygen increases. All these disturbances in

internal or external environment elicit an increase in ventilation, which serves to compensate for the original disturbance and thus protects the organism. The identifying characteristic of such compensatory adjustment is the increase in the ventilation equivalent for oxygen.

We may summarize the answer to our first question by saying that the functions of respiration are to meet the demands of the whole organism for carrying on its metabolism and to meet demands for compensating for certain environmental threats to its existence.

The anesthetist must be continuously concerned with the adequacy of the patient's respiration in meeting these demands of the body. True, nothing can be done about the metabolism of the patient, for it is fixed by the patient's own cells and tissues. But the pulmonary ventilation may readily become maladjusted, and interference with alveolar transfer may develop. These must be watched for and corrected or counterbalanced. Our second general question, therefore, is concerned with the ways in which respiratory processes may be disturbed and how these disturbances may be detected.

Disturbances of Respiration

Let us first consider maladjustments of pulmonary ventilation, and, for the sake of simplicity, only those involving maladjustment to metabolic requirements. The two kinds of such maladjustments are inadequate ventilation for metabolic requirements, known as hypoventilation, and excessive ventilation, or hyperventilation. From what has already been said, it should be clear that such maladjustments should be revealed by changes in the ventilation equivalent for oxygen. If the metabolic requirements are being met, the ventilation equivalent should be at its normal value of 2.5 L. of air for each 100 cc. of oxygen absorbed. A higher value signifies hyperventilation and a lower one hypoventilation.

The chief problem is hypoventilation, which may occur in a number of ways. Of primary importance is depression of the respiratory centers from excessive anesthesia or from any other cause. Likewise, excessive resistance to air flow, either in the anesthesia apparatus or in the patient's airway, will produce hypoventilation. Furthermore, excessive respiratory dead space in the mask or tubing will result in rebreathing of expired air. Normally, this will augment the total ventilation and thus restore adequate ventilation of fresh as distinguished from re-breathed air. But if breathing is depressed, or under control by the anesthetist, hypoventilation will result unless augmented breathing is induced.

Hyperventilation is less common, except when respiration is artificially controlled by an overenthusiastic anesthetist. The consequences of hyperventilation are less dangerous also. Hyperventilation does not interfere with oxygenation, but it does cause the body to lose carbon dioxide, a condition known as acapnia, and does increase the hydrogen ion concentration of the blood to produce alkalosis. The old notion that acapnia is dangerous and a cause of shock is now known to be false. Acapnia per se is not known to be dangerous; in fact it is Nature's own remedy for compensating for certain types of disturbances. Alkalosis, if severe, is the source of danger, because of increased neuromuscular excitability and tetany.

We have indicated that these maladjustments of ventilation are revealed by changes in the ventilation equivalent for oxygen, but facilities for continuous measurement of ventilation and oxygen consumption are rarely available. Perhaps the best that can be done is to recognize that the anesthetized patient must receive at least 5-7 L. of air a minute, depending somewhat on size, sex, and dead space of the apparatus.

Interference with the transfer of gases across the alveolar wall results from local pathologic conditions in the lung, such as pulmonary edema and atelectasis, which impair oxygenation of blood traversing the lungs. It may appear surprising that such interference does not reduce the total oxygen consumption until respiration or circulation is arrested. The explanation of this paradox is that even a fatal decrease in oxygen consumption by the brain, which is first affected, is scarcely detectable in the continuing oxygen consumption of the entire body. The poorly oxygenated blood may fail to support the vital regulatory centers of the brain, but oxygen consumption elsewhere will continue. The result is that interference with alveolar gas transfer is not betrayed by a fall in oxygen consumption but rather by a fall in arterial oxygen saturation. The clues to such interference consist, first, of signs of a local pathologic condition of the lung and, second, of cyanosis. Determinations of arterial oxygen saturation would be valuable but, of course, are not available. The observation of cyanosis is a substitute but a most treacherous one, since it is difficult to judge and may be absent when the patient's condition is most serious. In overcoming such interference with alveolar transfer, the anesthetist has an advantage over Nature. Nature can only resort to increased ventilation, which improves oxygenation only slightly and is apt to derange carbon dioxide elimination. The anesthetist, however, can increase the percentage of oxygen in the gas mixture; this is effective in improving oxygenation and does not involve any disturbance in carbon dioxide levels.

CONSEQUENCES OF INADEQUATE RESPIRATION

Our third and final question was concerned with the consequences of inadequate respiration. We have touched on this question all along, but there is more to be said, particularly with respect to the effects of inadequate ventilation, or hypoventilation. This condition produces three important changes in the chemical composition of the blood. First of all, hypoventilation causes a retention of carbon dioxide in the body, with a resulting increase in the free carbon dioxide of the blood. This is called *hypercapnia.* Secondly, hypoventilation brings about a decrease in the oxygen saturation of the arterial blood. This is called arterial *anoxemia.* Thirdly, both the hypercapnia and anoxemia cause a decrease in the hydrogen ion concentration of the blood. This is called *acidemia.* The acidemia results from an excess of two kinds of acid in the blood. One is the excessive free carbon dioxide, which is essentially carbonic acid. The other is lactic acid, which is produced in excess by tissues that are not receiving sufficient oxygen. Acidosis due to both the volatile carbon dioxide and the fixed lactic acid is especially severe, since with this combination compensation is not possible for either one.

This trinity of blood changes, comprising hypercapnia, anoxemia, and acidemia, is the inevitable consequence of hypoventilation. It is properly called *asphyxia,* a word that implies interference with both oxygen uptake and carbon dioxide elimination. It is not simple *anoxia,* such as results from breathing air deficient in oxygen, for this is ordinarily accompanied by hyperventilation, acapnia, and alkalemia. It is something far worse than simple anoxia, because the retained carbon dioxide makes an important contribution to the disastrous effects of asphyxia.

In simple hypoventilation, uncomplicated by a pathologic condition of the lung that would interfere with alveolar transfer, the free carbon dioxide of the blood increases markedly before the oxygen saturation decreases appreciably. By the time cyanosis appears, the free carbon dioxide will have reached a dangerous level. If the picture is complicated by local lung disease, the oxygen saturation may decrease concurrently with the increase in free carbon dioxide, and the acidemia likewise increases more rapidly.

It has been traditional to attribute all the adverse effects of this trinity of blood changes to one of them only, namely, anoxemia. In fact these changes are often described as anoxia. There is no doubt that severe anoxemia can be and often is fatal. But one cannot afford to neglect the equally serious effects of severe hypercapnia and acidemia. It appears not to be generally recognized that hypercapnia is as distressing as anoxemia is symptomless. A rapidly produced, simple anoxia, in which the arterial oxygen saturation decreases to 60 per cent or below, may produce unconsciousness without distress, sensation, or even any premonition of danger. In striking contrast, the breathing of 10 per cent carbon dioxide, which produces hypercapnia without anoxemia, causes marked distress and apprehension and can be tolerated only for about fifteen minutes. Longer exposures and higher concentrations induce narcosis, anesthesia, respiratory depression, and circulatory collapse. The potential danger of acidosis is well illustrated by the characteristic coma that develops in diabetic acidosis when the hydrogen ion concentration decreases to 7.1 or 7.0. In asphyxia, therefore, the threat of anoxemia is bad enough, yet it represents only one third of the potential danger.

Hypoventilation always carries the threat of progressing to a *crisis,* in which breathing rapidly weakens and stops all together. This is especially true of hypoventilation induced by deep anesthesia. The reason for this is that carbon dioxide in the presence of depression of the respiratory centers not only fails to stimulate breathing but actually inhibits it. Carbon dioxide in moderate concentration is a well known respiratory stimulant, but in high concentrations it depresses respiration. The critical concentration at which this reversal of effect takes place becomes lower and lower as the respiratory centers become more and more depressed. Even 4 per cent carbon dioxide can produce an abrupt cessation of breathing when the centers are sufficiently depressed by anesthetic agents. But the centers can also be depressed by oxygen lack and by severe acidosis, so that all three factors may be present in the asphyxia of anesthesia. The result is that a vicious cycle may be set up in which anesthesia, anoxemia, and acidemia depress the center to the point where the hypercapnia be-

comes inhibitory to breathing. The weakening respiration in turn aggravates the anoxemia, acidemia, and hypercapnia. The vicious cycle terminates rapidly in complete apnea.

When these progressive changes are initiated, what measures are available to the anesthetist to restore the respiration? Reduction of the amount of anesthetic agents, of course, is necessary. Increasing the percentage of oxygen is beneficial, since it will correct the anoxemia, reduce the formation of lactic acid, and relieve the respiratory center of some depression. But both of these measures together may not be sufficient, for they cannot be expected to correct the hypercapnia unless they restore adequate ventilation. For correction of hypercapnia, the ventilation must be restored so as to permit the retained carbon dioxide to be eliminated. *This cannot be done by administering carbon dioxide,* for this may inhibit respiration and obviously will interfere with elimination of carbon dioxide. Artificial respiration, by whatever means are available, is the only method of increasing ventilation to promote the elimination of excess carbon dioxide. Correction of the vicious cycle of asphyxia, therefore, requires the lightening of anesthesia, increasing the percentage of oxygen, and mechanical support of ventilation without the use of carbon dioxide.

SUMMARY

Pulmonary respiration is normally controlled in such a way as to meet the metabolic and compensatory needs of the body as a whole. This process may be upset by maladjustment of ventilation or by interference with alveolar gas transfer, the consequences of which may be disastrous. The anesthetist must follow these processes closely in order to recognize deviations early and correct them. I trust that this brief review of Nature's objectives in controlling respiration and of the ways in which respiration may be disturbed will help you when you find it necessary to come to her aid.

My Gift

To the Agatha Hodgins Educational *Loan* Fund

I hereby donate $_____to the Agatha Hodgins Educational Fund, which is "designed to extend financial assistance to graduate nurse anesthetists in obtaining further education and training to become qualified instructors, or to instructors who desire and need additional training to become better qualified."

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BALANCED ANESTHESIA AND SUPPORTIVE THERAPY

A meeting of the Institute for Nurse Anesthetists convened at 9:10 a. m., Friday, February 17, 1950, in the South Ballroom of the Stevens Hotel, Chicago, with Dr. Charles T. Dolezal presiding as chairman.

Chairman Dolezal: Our faculty this morning is from the Mayo Clinic, and I am going to ask Miss McQuillen to introduce them.

Florence A. McQuillen: Earlier this week one of the members of this institute made the remark, not to me, that it was wonderful that I could get away from my job so often to attend meetings as I have the last two or three years. They thought the Mayo Clinic was a wonderful place, and so do I. I am no longer with the Mayo Clinic, but it was my privilege to work with Dr. John Lundy and Dr. John Waugh. I now present Dr. Lundy.

Dr. Lundy: Members of the Institute, Miss McQuillen, Dr. Dolezal: The purpose of this meeting, as I understand it, is for us to attempt to say things that will be useful in your own environments. There are obviously a number of different circumstances under which you carry on your work, and I shall try to bear that in mind.

The subject this morning is balanced anesthesia and supportive therapy. We have come a long way in the choice of anesthetics. During my first three years at the Mayo Clinic one spinal anesthetic was given, and most of the anesthesias were by open drop ether. We had an old Ohio Monovalve that was built to administer nitrous oxide and oxygen as well as to get it out of the cylinders so that the company could sell more gas.

The first use of the expression balanced anesthesia was in 1925 or 1926 when I thought I could see the day coming when we would be putting part of the burden of anesthesia on premedication and part on a combination of anesthetic agents. Perhaps the best example of balanced anesthesia as it is used today is the administration of a drug such as nembutal or pentobarbital the night before and the morning of operation, the use of morphine or demerol by hypodermic injection with atropine before the operation, and then the administration of pentothal sodium and curare and half nitrous oxide and half oxygen, with or without intratracheal intubation. It has taken a great many years to develop this type of balanced anesthesia, and there is much to be said for it.

There are situations, of course, in which balanced anesthesia is not indicated. An example is abdominal or vaginal cesarean section. Pentothal sodium and the other anesthetics pass through the placenta, enter the circulation of the child, and make resuscitation difficult. In eliminating the hazard for the child infiltration anesthesia is used along the line of incision so that the abdominal wall may be opened and the wall of the

uterus exposed; then with only a few moments remaining to incise the wall of the uterus and remove the fetus, pentothal sodium may be administered, since very little of it will enter the fetal circulation. Almost every child delivered in this way cries the moment the head is born. For this type of anesthesia the patient must be rather stoical, for there seems to be hypersensitivity in the pelvic region of a woman at term or in labor. This is true whether continuous caudal anesthesia or abdominal block anesthesia is produced. When the caudal needle is put through the hiatus sacralis, an obstetric patient complains of more pain when the wheal is raised than does a surgical patient. The obstetric patient also resents the infiltration of the abdominal wall. To prevent this distress nitrous oxide and oxygen may be given in analgesic mixtures only.

Since curare does not pass the placental barrier, curare may be given for cesarean section without affecting the respiration of the child. Curare affects the end plates where the nerves meet the muscles and paralyzes this juncture, so that the nervous impulses have to jump a spark gap to activate the muscle, and in inactivated muscles the relaxation is profound. Syncurine (C-10), decamethonium bromide, has an entirely different pharmacologic action from that of curare. We are not certain whether it passes the placental barrier or not. If it does, its use in cesarean section would be contraindicated, because as yet no antidote for it has been prepared. In my experience with the use of the substance to produce relaxation in surgical anesthesia, the administration of more than 5 mg. to the average individual may result in inadequate ventilation. By inadequate ventilation I mean that, although the patient makes a feeble effort to breathe, the effort is insufficient to ventilate the lungs properly. If 10 mg. or more of syncurine is given, the next two hours may be spent in squeezing the bag and giving oxygen. Finally, of course, the drug is destroyed, and adequate respiration returns.

Curare, in my opinion, is preferable to syncurine, for curare has a physiologic antidote, prostigmine. It is advisable to administer prostigmine slowly and not to give more than 2 or 3 cc. of the standard solution, since cardiac depression may occur from an overdose of it.

With the extensive use of curare the popularity of spinal anesthesia has decreased. However, to preserve present day standards of education and practice, a variety of methods of anesthesia should be used in a hospital.

Briefly, balanced anesthesia is the use of a variety of agents, each carrying part of the burden of controlling pain. A parallel is found in a balanced diet, where one eats small or large quantities, whichever it may be, of several foods, rather than trying to survive on only one food.

With respect to supportive therapy there was a time when there were only a few solutions to be used, most of them being given by rectum. In the patient's room there used to be a crock containing a bottle of solution and a syringe to get the fluid out of the jar and into the patient's rectum or under the skin or under the breasts. There were

no stands to hold up a bottle and no handles on the bottles so that they could be turned upside down.

Now a variety of solutions of crystalloid material, such as glucose and saline, or sodium chloride, are readily available for administration by rectum, subcutaneously, intramuscularly, or, as a rule, intravenously.

Then came the day of the direct blood transfusion, and finally in 1915 sodium citrate was introduced to prevent blood from coagulating. The present vogue is to use citric acid, sodium citrate, and dextrose as the ideal vehicle for administering blood, so that it will not coagulate and may be preserved for three weeks when chilled to about 4 degrees above freezing.

The so-called plasma substitutes, and there are a number of them, include dextran—not dextrin, which is a starch, and not dextrose, which is a simple sugar. Dextran is a large molecule polysaccharide that is a nuisance in the beet sugar industry, where it plugs the pipes in the factories. Van Veglin in 1878 discovered that the sugar slime became infected with *Leuconostoc mesenterioides*, which is a standard organism. It grows in the sugar slime until the molecules have a weight of two to three million. They have to be broken down by hydrolysis, by acetone, and by straining through cellophane and centrifuged to remove the larger particles so that anaphylactoid types of reactions due to particle size will not occur.

Another plasma substitute is periston, the discovery of Weese, formerly of Cologne, Germany. The molecule is not very large, but when used in 3 per cent solution, it has the same colloidal osmotic pressure as plasma. Dextran used in 6 per cent solution in normal saline also has the same colloidal osmotic pressure as plasma. Periston in 2½ per cent solution was given to some 100,000 Wehrmacht soldiers by the Germans during World War II; this solution is hypertonic and not very satisfactory. I have tried it. I have also tried a 3 per cent solution, and I have finally realized that the 3½ per cent solution is the proper one to use. It is a little hypertonic, increases circulating volume in the cardiovascular system, and increases blood pressure.

In this category are also included gelatine, pectin, isinglass, and so forth.

I want to emphasize here that these materials for maintaining circulating volume in the cardiovascular system are not substitutes for blood or plasma. They have the same relation to a blood cell that water has to a boat. If the red cell is going to carry something, it needs something to float in. If the heart is going to circulate fluid, it needs fluid to produce the circulation. However, if a patient needs blood, because of hemorrhage or a reduced hemoglobin, he needs blood. He needs blood to aid healing processes, to help him recover from the anesthesia and operation as early and as completely as possible.

The use of so-called plasma substitutes is indicated when the hemoglobin is within normal limits, when there has been little or no loss of

blood, and when the blood pressure has decreased because of shock. For example, a patient is anesthetized with a general anesthetic in the upright position. In this situation there is a tendency for the blood pressure to decrease even though the legs are wrapped and other efforts made to support the blood pressure. Then the administration of a substance such as dextran will cause an increase in blood pressure.

Another difference between plasma substitutes, or any solution of crystalloid material, and plasma or blood used in supportive therapy is that the blood and plasma pass back and forth through the walls of the blood vessels into the tissues. This passage is extremely rapid, and a large quantity of blood and/or plasma may be administered without causing much increase in blood pressure.

I have a very unscientific attitude toward the use of blood in the presence of prolonged severe hemorrhage. In giving a transfusion to a patient who is, so to speak, bled out, some response is obtained from the first pint or two of blood and no further improvement from the next five or six or seven pints of blood; however, with the administration of the next pint, there is sudden improvement that is maintained. I think the spleen steals the blood. So far as I am concerned, the spleen is a reservoir of blood, and the tissues supply the fluid. It seems to me that, when the first two bottles of blood are given, spectacular improvement results because there is, for the moment, circulating blood when there hasn't been for some time. The spleen must then build up a reserve, and that is where I think those next few pints of blood go. Although I can find nobody to agree with me, the observation still stands that in treating hemorrhage a certain amount of blood is needed that does not appear to be beneficial.

Plasma substitutes, on the other hand, tend to fill up space, or to provide stuffing for the vessel. They tend to stay in the circulation. I cannot say factually that the spleen does not take them up, but I believe that the spleen has less affinity for these substances than it has for blood. In studies on shock it has been shown that, if a dog's abdomen is opened and the bowel massaged under ether anesthesia, the survival time is five or six hours less than if nembutal or phenobarbital is used as an anesthetic. Scientists agree that the difference in effect is due to the activity of the spleen. Ether causes the spleen to become smaller, and the blood is squeezed out of it. The barbitals cause the spleen to enlarge and store blood, and when the animal is in shock, there is an automatic transfusion from the spleen to the animal with a longer survival.

I am now going to tell you one thing that I am afraid I may forget to tell you. And if you will remember this one thing, I will feel satisfied with having come down here to make this speech. If you haven't seen a case like the one I am about to describe, you will—unfortunately. It is just a matter of time.

A patient who has been given an inhalation anesthetic ceases to breathe, for some reason or other, after having been cyanotic for some

time, and despite your efforts to get him pink again, his blood pressure decreases, his pulse is imperceptible, his heart seems to stop, and apparently he has ceased to survive. In desperation oxygen is administered, perhaps the heart is pricked with a needle, or perhaps adrenalin is injected into it. Then the heart suddenly begins to perform, and the pulse can be felt. The patient becomes pink again and finally resumes respiration, and his blood pressure and pulse become almost normal. But he is decerebrate. Usually, such a patient survives twelve to twenty hours before the heart fails, but he does not recover consciousness.

Some time ago, one of my associates, Dr. Harry Seldon, treated one of these patients, who had survived five or six hours, with serum albumin, 100 cc. every three hours for several doses, and the patient made a complete recovery. This has happened more than once. I know of two almost identical cases in which the patient's life has been saved and his mentality has apparently not been impaired. The effect is thought to be due to alleviation of edema of the brain. If you are ever faced with this problem of cerebral edema and anoxia and utilize this treatment, you may know that you have done the best thing that can be done under the circumstances.

Miss McQuillen: Dr. John Waugh is a surgeon at the Mayo Clinic, and he told me a while ago that I had given more anesthetics for him than any other anesthetist he had ever had. Dr. Waugh.

Dr. Waugh: I had never thought about any particular anesthetist's giving more anesthetics for me than any other until I was thinking about this program on the way down with Dr. Lundy, and I realized that Miss McQuillen had been with me at St. Mary's in one room all during the war. There is no question in my mind that she has administered more anesthetics than any other anesthetist for me, and I will say that they were good.

Dr. Lundy gave you a good definition of balanced anesthesia. However, being a surgeon, I can think of one that appeals more to me: You have balanced anesthesia when the patient, the anesthetist, and the surgeon are all happy. I don't know of anything that can disturb the surgeon more than not having confidence in his anesthetist. No matter how dextrous the surgeon, if he doesn't have confidence in the anesthetist and isn't getting proper anesthesia, the operation and the result are probably not going to be satisfactory.

The question I have here is: **What is the cause of hiccoughs during gastric resection, and how should they be prevented and treated?**

I think that hiccoughs are caused by three factors: First, they may just appear, and no one may be responsible. The surgeon may be conducting himself properly, and the anesthesia may be satisfactory. Some patients just have hiccoughs, and there is nothing you can do about it but go ahead and put in a stitch between hics.

Second, the surgeon may be responsible for hiccoughs by exerting traction on the upper part of the stomach and esophagus. Frequently, if he will release the traction for a minute or two, the hiccoughs will disappear. The anesthetist may then deepen the anesthesia for that particular part of the operation. I think most hiccoughs during gastric resection would be obviated if the surgeon would give the anesthetist a chance to deepen the anesthesia. In most gastric resections, except during incision and closure, a great deal of relaxation is not needed. However, relaxation is needed during transection of the stomach, and at that point the anesthesia should be deepened for a few minutes.

The third factor is the anesthetist, who may cause hiccoughs by being a bit too avid in pumping the bag, with the result that air gets into the stomach. This may be readily taken care of with a tube in the stomach.

We used to be frightened when hiccoughs occurred during anesthesia, but today we do not believe the occurrence of hiccoughs is a reason for stopping the operation as long as the patient is in good condition.

What is the consensus with respect to the administration of spinal anesthesia by a nurse anesthetist?

The answer to that varies with the circumstances. However, if I were a nurse anesthetist, I would hesitate to give a spinal anesthetic. Custom, of course, determines the responsibility. Custom has determined that it is perfectly all right for the nurse anesthetist to give inhalation anesthetics, and the surgeon or the hospital assumes the responsibility. But at the present time custom has not determined that it is proper in all instances for a nurse anesthetist to administer a spinal anesthetic, and I grant that many of you no doubt could do it better than I could.

It has been my observation that the administration of demerol sometimes causes considerable respiratory depression in infants, the opinion of the manufacturers notwithstanding. What is your opinion?

As a general rule, we don't give any premedication to infants, except a small dose of atropine, usually gr. 1/300, unless the operation, for example, an operation for pyloristenosis, is being performed under local anesthesia. Then we give nembutal, gr. ½, rectally, and that with a little whiskey seems to do the trick with the local anesthesia. I think that giving morphine or codeine to the newborn is rather dangerous and is not needed. If something is needed when the operation is to be performed under local anesthesia, nembutal with atropine is very satisfactory.

What are the causes and treatment of acute dilatation of the stomach during an operation?

There are several possible causes. The stomach may not have been emptied by gastric intubation. However, the patient undergoing a

gastric operation usually vomits even though gastric intubation has not been done, so usually the stomach will be depressed although not collapsed.

Another cause is the pumping of air into the stomach by positive pressure on the bag. I have also seen patients get a swallowing reflex with terrific dilatation of the stomach. The treatment is to insert a tube and empty the stomach.

When an intratracheal tube is used to give oxygen after a thoracic operation, the tube may inadvertently be placed in the esophagus instead of the trachea. I know of two patients who have had rupture of the stomach due to increased pressure in the stomach. The tear is small, and the treatment is to decompress the stomach with a tube.

Is the administration of fluids intravenously during major operations recommended?

It is almost routine with us to have intravenous fluid therapy started while anesthesia is being induced and before the incision is made. Usually, 5 per cent glucose solution is used. However, if the patient has been vomiting and has lost chlorides, sodium chloride solution is used.

If the patient is in shock, obviously, intravenous administration of fluids is needed. If the patient's condition is such that blood will be needed, a transfusion should be started at the time the incision is made. In summary, I would say that for a major operation a vein should be cannulated routinely for the administration of 5 per cent glucose solution.

Miss McQuillen: Dr. R. C. Adams is known to most of you as the author of *Intravenous Anesthesia.* Dr. Adams.

Dr. Adams: The first question is: **Outline the treatment of laryngospasm.** I assume from the subheadings that this means laryngospasm associated with pentothal sodium anesthesia.

I think we have found that laryngospasm is not so serious or so common a complication of intravenous anesthesia as many have made out. When it is severe, it can be very serious and very annoying, but some go so far as to say that it is one of the major complications of intravenous anesthesia. We don't find it so. I think that if due attention is paid to the airway, if mucus is kept out of the throat, and if the surgeon is kept from beginning before anesthesia is sufficiently deep, laryngospasm may be prevented in many cases. The first step, when laryngospasm occurs, is to stop the operation and give oxygen under slight positive pressure; this treatment is usually sufficient to relieve the spasm. Often atropine, gr. 1/150, given intravenously and slowly will aid materially in relieving the spasm; often it will not. We prefer atropine to scopolamine for this purpose.

The spasm cannot be obliterated by deepening the anesthesia. Anoxia is already present, and if more pentothal sodium is given,

respiration is further depressed, the anoxia is increased, and nothing is done to relieve the spasm. Pentothal sodium does not obliterate laryngeal reflexes adequately; only after some time and considerable dosage can laryngeal reflexes or pharyngeal reflexes be very well obliterated. For that reason we do not consider pentothal sodium a good anesthetic for tonsillectomy. The reflexes in the throat are too active, by and large, to permit the use of pentothal sodium in what we consider to be safe doses.

During pentothal sodium anesthesia, and other types of general anesthesia as well, we have found that small doses of curare will often obliterate laryngeal reflexes and relieve spasm. This is not always true, and one should be cautious in determining dosage. If 20 units, or 3 mg., does not relieve the spasm, probably twice or three times that amount won't either.

I have already mentioned the use of oxygen under positive pressure. Possibly the addition of helium might be of some help, although we don't use it. We prefer to use oxygen alone. By using slight and intermittent positive pressure, one is usually able to get a little oxygen through the cords, and gradually the spasm is relieved.

I consider intubation to be the last resort in the treatment of laryngospasm. In many, many thousands of cases of pentothal sodium anesthesia we have rarely found it necessary to insert an intratracheal tube. However, in some cases that are difficult to control it is necessary to establish an airway. In other words, if the spasm is not relieved within two or three minutes and cyanosis becomes severe, an airway must be established by some means or other. Under these circumstances I think there is reason for forcibly inserting an intratracheal tube through the vocal cords. It may be traumatic, but an airway must be established. However, as I say, it should be considered only as a last resort.

Is the use of pentothal sodium advisable in thoracic surgery?

Various thoracic surgeons have pet anesthetics for thoracic operations. I think the problem should be considered in the light of the essentials of chest surgery. One is a clear and unobstructable airway. Second, facilities should be available for the successful aspiration of the tracheobronchial tree if necessary. Facilities should also be available for giving positive pressure. A great deal of relaxation is not needed for a thoracic operation, and therefore the more potent anesthetic agents are not required. All that are needed are moderately depressed respiration and a quiet chest. Accordingly, although we do not use pentothal sodium anesthesia routinely for thoracic operations, since our thoracic surgeons tend to favor ether, I do not see any particular contraindication to its use, provided the fundamental requirements mentioned are met.

Another important factor in thoracic surgery is the insertion of a large needle in a vein for the administration of fluids. Complications

arise quickly during open chest operations, and preparations must be made to give supportive therapy without delay.

In summary, if an intratracheal tube is used—and it should be for any thoracic operation regardless of the type of anesthesia used—, if the facilities mentioned are available, and if the amount of pentothal sodium administered does not unduly depress respiration, pentothal sodium may be used with reasonable safety. If overly large doses seem to be required or if the respiration becomes unduly depressed, it is wise to switch to one of the old standbys such as ether.

What is the choice of anesthesia for a patient with diabetes?

In the old days, before internists had diabetes so thoroughly under control, the choice of anesthesia for a patient with diabetes was entirely different from what it is today. Then the anesthetist had to worry about variations in the blood sugar and about anesthetics that would affect the blood sugar level. Now the patient is so well prepared preoperatively that it matters little what type of anesthesia is used. Spinal anesthesia or pentothal sodium, both of which have little effect on the blood sugar level, is the method of choice. Although ether affects the level of the blood sugar considerably, it may be used safely if the patient is under the care of a competent diabetic specialist.

What is the choice of anesthesia for a patient with cardiac disease?

In my opinion, if a patient is able to walk to the operating room, lie down in bed without being propped up with two or three pillows, showing that he has no cardiasthenia, and carry on a fairly normal life —I don't mean strenuous activity—, his heart will probably tolerate almost any type of anesthesia. However, the choice is probably between local or regional anesthesia if the operation is minor and ether if it is major.

It is important in anesthetizing a patient with cardiac disease to see that the heart is adequately supplied with oxygen no matter what type of anesthesia is used. A damaged heart, particularly a damaged myocardium, will not tolerate anoxia for any length of time or to any severe degree. If the anesthetic is cyclopropane, spinal, or ether, the patient must be assured an adequate supply of oxygen to take care of the cardiac function. This, I believe, is the most important factor in anesthesia for a patient with cardiac disease.

What is the anesthesia of choice for a patient with tuberculosis?

You have heard a lot about the irritating effects of ether on the lungs of a patient with active pulmonary tuberculosis. I think that theory has been largely exploded. Possibly, when local, regional, or spinal anesthesia or an agent like pentothal sodium that does not produce mucus and probably does not irritate the tracheobronchial tree can be used, it is to be preferred. However, ether, according to many leading thoracic surgeons and anesthetists, is not contraindicated in tuberculosis.

What is the anesthesia of choice for very old patients with diabetes and cardiac disease?

We have covered diabetes and cardiac disease. For very old patients ether should probably be avoided. Certainly, the extremes of age are no contraindication to the use of pentothal sodium provided it is given cautiously and in doses suitable for debilitated, frail, elderly people. An old person will tolerate pentothal sodium just as well as a husky young adult, but the dosage required to anesthetize him will probably be a quarter or less of what the average adult would take.

What is the choice of anesthesia for a patient with asthma?

We don't pay much attention to anesthesia for the asthmatic patient. Some say pentothal sodium is contraindicated. There have only been a few cases in which I have thought that asthma was aggravated by the administration of pentothal sodium, and, personally, I do not consider it a definite contraindication. Local, regional, or ether anesthesia may be used with safety.

What would be the best method of induction of anesthesia for tonsillectomy in a patient having asthma?

The question does not state whether the patient is an adult or a child. Whenever possible, we believe that tonsillectomy in an adult should be done under local anesthesia. The margin of safety, I think, as far as postoperative pulmonary complications are concerned, is much wider when local anesthesia is used. This would be true of an adult whether he had asthma or not.

For a child with asthma, or for any child undergoing tonsillectomy, induction with a little nitrous oxide and maintenance with drop ether, either with or without intratracheal intubation, are satisfactory.

At the Clinic intratracheal intubation is used for most children undergoing tonsillectomy. After we had used intratracheal intubation for a while, the surgeons found that the airway was much better, that there was less mucus, and that the tube could be retracted from one side to the other without interfering with the operative field. When the operation is finished, the child is turned over, the intratracheal tube taken out, and the child left on the operating table, his head held so that blood and mucus can run out of the throat and nose. The child is left there until he vomits and is essentially awake. I think this procedure insures a safer and more trouble-free postoperative period. When the child goes to his room, even though he is awake and has vomited and got rid of the blood and mucus in the throat, he is still left in the prone position with the head turned to the side.

What is the safest anesthetic for enucleation?

It depends on how long the surgeon takes to do the job. Many surgeons can take out an eye in a very short time. Since no general relaxation is needed, it seems unwise to fill the patient up with ether. Accordingly, I think pentothal sodium is the anesthetic of choice. If

the surgeon is going to take considerable time or if complications are anticipated, an intratracheal tube should be inserted. Otherwise, after the needle is inserted and the tube hooked up, the anesthetist can grasp and support the jaw and in most instances maintain an adequate airway.

One of the complications of pentothal sodium anesthesia for enucleation is sneezing. It doesn't occur too often, but when it does, it can be a real complication. The anesthesia may be progressing smoothly when, all of a sudden, the patient will heave, and if the surgeon isn't careful, the instrument may go through the eyeball. This does not refer only to enucleation but to any kind of operation on the eye. Presumably, the sneezing is caused by irritation of the lacrimal duct. The only way to prevent sneezing is to have the surgeon wait until the anesthesia is sufficiently deep to obtund the reflexes.

Do you advise preoperative medication for babies?

The question does not say how old, so let us take the age group from 1 to 3. I think an opiate is unnecessary and probably dangerous. I doubt if morphine should be given to a child under 10 to 12 years of age, although if given in very small doses, it probably is all right. However, for many children an opiate is hardly necessary. Even very small doses of morphine cause depression in infants. So we prefer to avoid morphine. Sometimes codeine, gr. ½, is given in its place. Even this I don't think is necessary.

If the child is 6 months to 1 year of age, we usually give a small dose of atropine, although sometimes we give no atropine at all. To a child, aged 6 months, we give atropine gr. 1/400 and gradually increase the dose according to the child's age. To a child of 4 we give the full dose of gr. 1/150. Most children are underatropinized rather than overatropinized.

We also give a barbiturate, preferably by rectum. A child of 3 will tolerate nembutal, gr. ¾ to gr. ½, by rectum. The sedation is fairly satisfactory, and I think possibly that is all the preliminary medication that a baby needs.

What is the anesthetic of choice for an adult scheduled for tonsillectomy?

If general anesthesia is to be used, the scheme of preliminary medication should be the same as that for any other type of operation. We give nembutal, gr. 1½, the night before and sometimes gr. 3 if necessary. It depends on the patient's emotional status. If the patient is high strung, more sedation will be required. It must be remembered that the anesthesia is started with premedication the night before operation. It is desirable to size up the patient and give him the amount of preliminary medication it takes to make him dopey, drowsy, half asleep, but not depressed, by the time he reaches the operating room. That is the ideal level of preliminary medication. About an hour before

the patient is to go to the operating room, he receives nembutal, gr. 1½. If the patient is asleep when you go into his room, additional nembutal probably is not necessary. However, if he is wide awake, alert, and worried, nembutal, gr. 1½ to gr. 3, may be given. Each patient must be individually appraised. Probably nembutal, gr. 1½, would be the right amount. About one half hour before the patient goes to the operating room morphine, gr. 1/6, should be given. That is our routine. I think many patients would do better with morphine, gr. ¼, if they are high strung. Atropine, gr. 1/150, is also given.

DR. LUNDY: There are times when you need to become a gadgeteer, and I think most persons in anesthesia like gadgets. It seems to go along with the desire to administer anesthetics. So I am going to break away from these questions for a few minutes and talk about gadgets.

There are times when an anesthetist is faced with bizarre situations. Sometimes there is only one vein left for venipuncture, or whoever is to do the venipuncture may be unable to get a needle into any but one vein. We put a 17 gage needle into the vein, and through it we thread polythene tubing (fig. 1). I have a little here, and I will be glad to give you six inches of it, which is enough for one case. It is the type of material that cannot be boiled. It is sterilized by cold sterilization in any ammonium base antiseptic for ten hours.

You will note that there is a bevel on the end of the polythene tube. That bevel should be as short as possible so that it won't plug up. After the tubing has been inserted into the vein, the needle is withdrawn, the tubing is left in place, and a small turn or knot is made in it. I am sure you have seen the stunt where somebody puts a cigaret in a plastic tube and ties the tube in a knot without breaking the cigaret. That is the idea of this tubing. This tubing is tied in a loose knot because you can't get hold of the tubing with the adhesive unless there is something to take hold of. Then on the end of the tubing is placed a small bore needle. A cap may be screwed on the end of the needle to keep it clean. If you are going to leave the tubing in the patient for two or three days, the floor nurse should be instructed to inject 2 cc. of normal saline solution into it every two hours. Solutions may be dripped through the tubing, but blood does not drip through it well. However, blood may be given by putting a valve on the needle with a syringe on it to make a two-way valve. The blood is drawn into the syringe and then forced through the tubing into the patient.

This appartus comes in handy for certain small patients, for example, a child with congenital malformation of the biliary tract who is to undergo a long difficult operation. The 17 gage needle is inserted into the vein as soon as the anesthetic is administered, for at that time, before the operation is started and after surgical anesthesia has been produced, the veins assume their greatest diameter. Otherwise, heat must be applied to the extremity to make the veins dilate as much as possible. The 17 gage needle is left in place during the operation, and

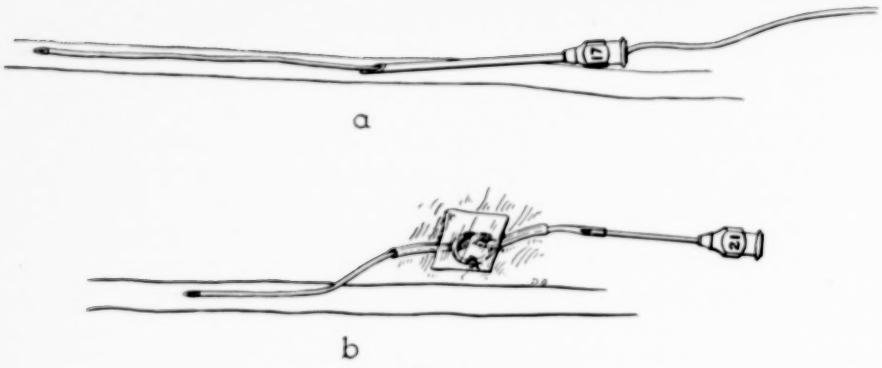

Fig. 1

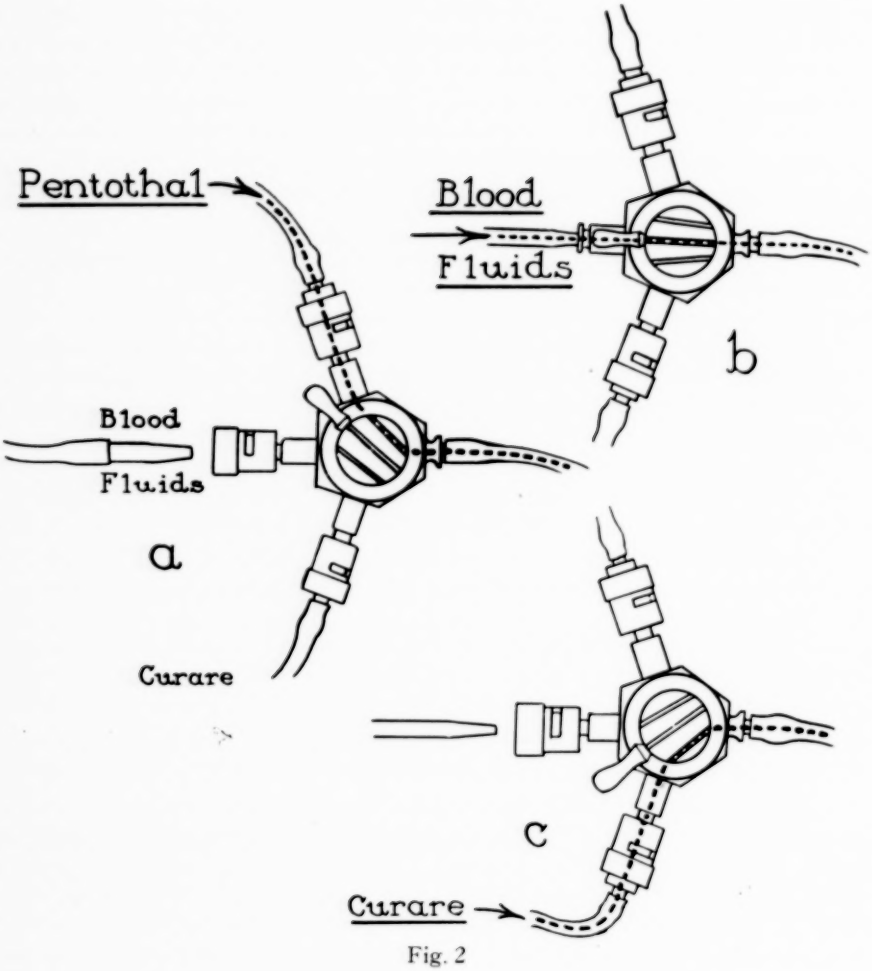

Fig. 2

through it blood is allowed to drip by the gravity method. When the operation is over, the tube may be inserted through the needle, the needle removed, and a small bore needle placed on the end of the tubing. Thereafter, it is a simple matter for anyone to administer fluids night or day by simply unscrewing the cap on the needle.

At the Clinic we have a gadgeteer, by the name of Dr. Massa, who has devised what he calls a polythene needle. The needle is on the inside of the tubing and extends beyond it. As the needle is forced into the vein, the tubing follows. The needle is then pulled out. This arrangement is better than the one just described in which the needle, being larger than the tubing, makes a larger hole in the vein than the tube. Consequently, leakage may occur if the tubing is retracted toward the hole.

We are also working on a device to hold the needle so that it will not move. If the Massa needle moves back and forth through the hole in the skin, infection will result. Also if there is some pull on the needle, it tends to pull the tubing close to the hole in the vein to make it easier for the fluid to leak out than to run through the vein.

We like to give pentothal sodium and curare separately, although some anesthetists like to mix it together. I do not care to mix curare and pentothal sodium together any more than I would like to mix oxygen and nitrous oxide in one cylinder on the gas machine for the purposes of simplicity.

The valve for this gadget for giving pentothal sodium and curare separately was made for us by Becton, Dickinson and Co. (fig. 2). The valve may be turned without reflex flow up the tubing to the container. There is very little mixing between the valve and the needle. Because both hands were needed to work the gadget, we had a little clamp made that goes on the table, and to it is attached a little rod topped by a ringlike setting and a little turret, in which the valve is set so it can be turned with one hand.

A little while ago I spoke about the patient who becomes anoxic and decerebrate. Other patients who have lesions of the brain need to be given artificial respiration for some time. Recently, we had such a patient upon whom I performed intratracheal intubation. The next morning I was called because the patient did not seem to be having adequate ventilation. On removing the tube I found that it was plugged up at one end with secretions. So it is important to remember that in such a case the tube may become plugged up if left in place very long.

What drug companies distribute dextran, periston, and so forth?

I shall start with gelatin because it isn't mentioned here. The Upjohn Co. of Kalamazoo is about to put a very fine gelatin product on the market. Winthrop Chemical Co. has gelatin prepared for intravenous use. The Knox Gelatin Co. has a gelatin product that can be used.

A liter of gelatin of about 40,000 molecular weight is very satisfac-

tory for maintaining circulating volume. For example, if 1,500 cc. blood were needed, we would probably give 1 L. blood and 1 L. gelatin and get the same result as we would with 1,500 cc. blood.

Periston, as I told you, is obtained from British-occupied Germany. Recently, the General Analine and Film Co. in this country discovered that it could make periston. The National Drug Co. of Philadelphia has sent me a concentrate, and the Abbott Laboratories of Chicago has sent me some solutions containing polyvinyl pyrolodon. But, as far as I know, periston is not yet commercially available.

Dextran, by the way, comes in 500 cc. bottles. The cost to the hospital is $10 a bottle f.o.b. Rochester. On the other hand, we pay $25 a bottle for blood and $25 for the administration of the blood, and the hospital gets $2 for the transfusion, totaling $52.

How much serum albumin do you recommend for administration?

The Cutler Laboratories makes serum albumin, and other companies may also. In the very severe case of cerebral edema, 100 cc. is given every three or four hours for five or six doses. So far as I know, the lowest price for serum albumin is about $1 a cubic centimeter. However, if serum albumin will perform the miracle that I have seen it perform, the price isn't very important. There is no discarding date limit on serum albumin, and we don't pay any attention to how old it is.

What is your opinion of the administration of 5 per cent alcohol glucose solution after operation?

It is a rather good preparation to use when the circulation is inadequate and after operations performed on blood vessels, especially after operation for aneurysm.

The solution is 5 per cent alcohol in 5 per cent dextrose normal saline solution, or 5 per cent alcohol in normal saline solution, or 5 per cent alcohol and 5 per cent dextrose solution. It all depends on whether the patient is a diabetic or not and whether the diabetes is easily or not easily controlled. If the patient is a diabetic, it is wise to get the opinion of the diabetic service about administration of the solution. If the solution is given too fast or if more than 3 L. a day is given, the floor nurse will have trouble with an inebriated patient. Alcohol glucose solution does increase peripheral circulation.

What is the cause of a slow pulse in an elderly patient having extensive perineal repair or hysterectomy under pentothal sodium anesthesia?

Dr. Adams: Bradycardia during anesthesia, whether the agent is pentothal sodium or any other agent, probably isn't due to the agent itself. Of course, it depends on whether anesthesia is being maintained with a closed system. Sometimes carbon dioxide accumulation will cause bradycardia. I don't believe the particular type of lower abdominal or perineal operation would have anything to do with it. I don't think pentothal sodium in itself would particularly have anything to do with it, because pentothal sodium ordinarily does not affect the pulse rate too much.

DR. LUNDY: I think it must be perfectly obvious, if you have lived as long as I have, that anything can happen. Also, curiously enough, the environment in which you work is always quite different from that of someone else. I go around the country and visit operating rooms, and I see certain things happen that I have never seen at the Clinic. How do you account for it? You have to go back to the preliminary medication and the type of operation. No two surgeons, as far as I know, do vaginal hysterectomy with the same sort of traction, and respiration can be affected by dilating the anus, by traction on the gallbladder, by trying to get a fibroid out of the pelvis, or by abdominal exposure. I would like to have Dr. Waugh explain that.

DR. WAUGH: I certainly believe that the surgeon would have to assume some of the responsibility. I prefer spinal anesthesia for vaginoplasty and vaginal hysterectomy mainly for one reason. That is that I have found that less blood is lost. I have used everything else—ether, cyclopropane, and pentothal sodium and curare. But I am satisfied that the way I do vaginal hysterectomy less blood is lost when spinal anesthesia is used.

Interestingly enough, most women don't want spinal anesthesia. Occasionally, one will ask for it. Many men ask for a spinal anesthesia. Since I dislike to have a patient take anything she doesn't have confidence in, I use nitrous oxide and ether if I can't use spinal anesthesia. I also like to have the patient as deeply anesthetized as she would be for cholecystectomy. There will be less bleeding.

I don't know why bradycardia would occur, because it hasn't in our experience, as Dr. Lundy indicated. However, I believe that the surgeon has a good deal to do with it, and the reason is not necessarily loss of blood. Traction may be the cause. Certainly, with spinal anesthesia the patient will have pain when the round ligament is retracted in the latter part of vaginal hysterectomy, and frequently a slight decrease in blood pressure will occur. It is almost routine with us to give 1,000 cc. blood during vaginal hysterectomy. We have found that blood loss is between 500 and 800 cc., so almost every patient needs 500 cc. as a minimum. Blood loss is also less when spinal anesthesia is used.

DR. ADAMS: I don't know that any of us has been too helpful in answering this question. Probably the most reasonable explanation is, as Dr. Lundy stated, that the bradycardia is due to stimulation of reflexes at the operative site. As he said, that can affect respiration, and it can affect pulse rate, and so forth. I think it is reasonable to assume that traction in this instance would probably cause the slow pulse.

What is the cause of jactitation or twitching during pentothal sodium anesthesia?

It might be due to a number of factors. Jactitation occurs quite often during induction. Fortunately, it does not occur very often under pentothal sodium anesthesia. The cause may be inadequate prelim-

inary medication, slow prolonged induction, or the position of the patient on the table. If a leg is poorly placed so that some of the muscles are tensed, reflex twitching or jactitation may occur. Fortunately, induction with pentothal sodium is so rapid that jactitation is rarely seen at that time. It is seen in a moderate number of cases during recovery. I think it is an excitation phenomenon, and it is not serious. It usually requires no special treatment.

Is there any indication for the rectal administration of pentothal sodium?

By and large, we are against the administration of barbiturates, except nembutal, by the rectal route. When a barbiturate is given by rectum, the control that is possible by intravenous administration is lost. In other words, the total dose must be given at one time, and one cannot be sure of the patient's tolerance to the barbiturate. Everyone has a different tolerance to barbiturates as he has to alcohol. For this reason pentothal sodium is given intravenously. A test dose may be given, 1 or 2 cc., and from the reaction to the test dose an estimate made of how much the patient will take. If 2 to 4 cc. of the 2½ per cent solution of pentothal sodium produces moderate narcosis, the patient probably requires a very small total dose of pentothal sodium for the operation, and subsequent administrations should be made cautiously. If the patient requires 10 to 20 cc. for induction, as frequently happens, he has a high tolerance. Because we believe such fine control is possible by giving pentothal sodium intravenously, we see no particular point in giving it by rectum in most instances.

Dr. Waugh: **Do you use dextrose in distilled water in preference to dextrose in normal saline solution?**

I might say, "Yes," because the average patient does not need additional saline. If there has been vomiting or loss of bile from a fistula, or if the patient has a deficit that can be corrected by giving saline solution, it is administered, but as a general rule 5 per cent glucose in distilled water is the solution preferred for intravenous administration during a major operation.

Do you give blood routinely during upper lumbar thoracostomy?

Usually, there is no appreciable blood loss, and the problem is to combat a decrease in blood pressure. The use of dextran is ideal for this purpose, and I imagine that more dextran has been used at the Clinic after this particular procedure than after any other. In other words, in the absence of blood loss but in the presence of a decrease in blood pressure, dextran seems to be the ideal agent for intravenous therapy.

I would like to describe the case of a patient on whom I operated last November or December. She was a young woman from Georgia, who was referred to me by her surgeon there. She had been operated on several times for the correction of an intestinal fistula that followed peritonitis after pelvic laparotomy. The womb was filled with adhe-

sions, and stripping the bowel free from the adhesions produced shock. She was given five or six pints of blood because the adhesions were bleeding freely. Within a few hours we gave her seventeen bottles of blood without correcting the shock. Then two bottles of dextran of 380 cc. each were administered, and her blood pressure became normal and stayed normal. I was sold on dextran from there on, and I think we wasted a great deal of blood. She had 18 Gm. hemoglobin, so she didn't need all the blood we gave her, but we thought she might be bleeding in the peritoneal cavity although there were no signs. When more blood is being given than the patient has lost without correcting his condition, it is wise to switch to dextran.

What type of anesthesia would you recommend for an unconscious patient on whom decompression needs to be performed?

No anesthesia may be needed. Usually, local anesthesia will be sufficient, because an unconscious patient should be given no more of an anesthetic than is necessary. Frequently, the unconscious patient after decompression will begin to wiggle a foot or leg and begin to talk, and that is what the neurosurgeon wants. If necessary, a very small amount of pentothal sodium may be given if the patient is wiggling so much that the anesthetist can't hold him.

What anesthetic agents and technics are best tolerated by accident patients in poor condition?

As a general rule, the patient is in shock, and I would say that spinal anesthesia and pentothal sodium are contraindicated. If possible, local anesthesia should be used. If not, a general anesthetic is safer than the other two agents as a rule. However, there are instances in which spinal anesthesia may be used, although one should beware of large doses of the agent because of shock.

What anesthetic should be used when cautery is being used for coagulation?

If cautery is used routinely for certain operations, a nonexplosive anesthetic should be chosen. If I were in the habit of using cautery, I would ask for spinal anesthesia or pentothal sodium and curare. However, if the gas machine is moved out of the operating room and the anesthetic switched to open drop ether, there should be no problem.

What is the preferred anesthetic for lobectomy or major chest surgery without the use of cautery?

DR. LUNDY: We have made a number of efforts to arrive at the anesthesia of choice for lobectomy. The surgeons have their opinions, and we have ours. In general, our thoracic surgeons seem to believe that nitrous oxide-oxygen and some ether with an intratracheal tube is a satisfactory combination. I believe that there is hardly any general or thoracic operation that cannot be done under pentothal sodium, curare, nitrous oxide, oxygen anesthesia with the use of an intratracheal tube. I am particularly fond of it because it is explosion proof and the patients

like it. Nevertheless, generally speaking, my advice would be to use nitrous oxide and oxygen and ether with an intratracheal tube for lobectomy.

What is the reason for an increase in blood pressure with the administration of pentothal sodium alone or pentothal sodium and oxygen, usually in Negroes?

It is a little difficult to detect cyanosis in certain Negroes, and cyanosis is always attended by an increase in blood pressure, as is carbon dioxide accumulation. Although in Rochester we do not have many Negro patients, I understand some of them dread an operation, although that fear is not restricted to Negroes. If the patient is sufficiently excited during induction, an increase in blood pressure will occur, followed by a decrease in about fifteen minutes. The patient excretes adrenalin, which has an effect for about fifteen minutes, as compared with ephedrine, which has an effect for about forty-five minutes.

In this connection administration of a vasopressor substance is needed, more often with gelatin than with dextran. However, if the administration of dextran is accompanied by a slight increase in blood pressure, a vasopressor substance should also be administered. Although the results from the use of dextran are spectacular, it alone is not the complete answer in every case.

I would like to hear a discussion of the postanesthesia room.

We have operated a postanesthesia room in St. Mary's Hospital since March 17, 1942. About one bed for each operating room is needed. The patient stays in the postanesthesia room about as long as he does in the operating room. One in a thousand falls out of bed unless it has sideboards. Usually, one nurse and one orderly are assigned to the room and often two nurses and an orderly.

Before going to the postanesthesia room, every patient is seen in the operating room by the supervising anesthesiologist. The anesthesiologist checks the patient and the record to make sure that the record is complete and correct and for the right patient. The patient's relatives should be told in advance that he will go to the postanesthesia room unless he is sufficiently awake to go to his own room.

Several floor supervisors at St. Mary's have announced that they will resign if we close the postanesthesia room. When three or four patients from one floor are operated on at about the same time and are returned to their rooms immediately after operation, the supervisor has to let three or four nurses sit with those patients until they wake up, while she tries to take care of the rest of the patients.

What are the objections to starting blood transfusions with glucose solution?

When glucose solution is administered with blood that has been stored for some time, there is a tendency for blood cells to cluster

together. Since the clusters may escape the filter, you may be criticized for mixing the glucose solution and blood. Consequently, it is well, to avoid an argument, to disconnect the tubing at the needle and let the tube fill with blood.

May cold blood be used in an emergency?

Yes, cold blood may be used in an emergency. Also, there is no quicker way to cool a patient with hyperthyroidism than to give him cold blood. In the presence of shock it is sometimes difficult to get cold blood to run into the vein because of venous spasm. The blood vessels go into spasm in an automatic effort to maintain blood pressure. However, as the shock comes under control, the blood will suddenly start to flow freely, which is a good sign that venous spasm is letting up.

If the patient is in shock and the blood is dripping slowly, you may wonder if the blood is too cold, if the container is high enough, or if the needle is big enough. If you will constrict the limb about 18 inches above the needle and get pressure against the vein, the vein will dilate, and when the tourniquet and blood pressure cuff are released, the blood will flow freely. Pressure in the bottle may also be used to make blood flow faster.

Is it safe to use outdated plasma?

No.

What type of anesthesia should be used during x-ray examination?

The anesthetic should be fireproof and explosion proof, e.g., pentothal sodium or oxygen and nitrous oxide on a gas machine without an ether bottle. A gas machine that you are going to throw away may be adapted for use in x-ray examinations with a cylinder of nitrous oxide and one of oxygen and a bag and mask.

Do you use pentothal sodium for children at the Mayo Clinic?

DR. ADAMS: When pentothal sodium was first used, its administration was restricted to persons 10 years old or older. We also used very small doses, ¼ to ½ cc. of the 2½ per cent solution prior to transfusion to quiet the child. The limit was set because a comparatively small dose of pentothal sodium may be more depressing to a child than to an adult. We now believe that pentothal sodium may be used for comparatively young children, 5 or 6 years of age, if it is easy to get into a vein and if it is given very cautiously, slowly, and in small doses. By and large, it is not the best anesthetic for very small children.

MISS MCQUILLEN: The management wants us to be out of here promptly because this room has to be set up for our luncheon. We will have one hour. We will try not to impose on our speakers. They want to collect their wits and their wives to come back and have lunch with us.

NOTES

CASE REPORTS *are the foundation of the medical literature. Their brevity makes them interesting to write and assures their being read. There is a place in this section of* NOTES *for case reports as well as for descriptions of gadgets and special technics. Send in your contribution now. Other anesthetists will be helped by it.*

TWO CASE REPORTS ON ANESTHESIA AND SURGERY FOR LUDWIG'S ANGINA. — Ludwig's angina was first described by Ludwig in 1836. The description has not changed. It is a rapidly spreading cellulitis in the submaxillary region. As a rule, it is caused by a pure virulent streptococcic infection.[1] It usually follows the extraction or infection of a lower molar tooth. If the condition is unrelieved, pus burrows between the mylohyoid and middle constrictor of the pharyngeal muscles.[2] It begins as a hard swelling and may continue as such for several days, when glottic edema may ensue with dramatic suddenness. The tongue becomes very thick, speech is difficult, there is excessive salivation, the temperature as a rule is not high, and the patient becomes profoundly toxic.

The very word angina means strangling, and the mortality rate in Ludwig's angina is extremely high owing to strangling from acute edema of the glottis. Barnhill[3] stated that the mortality rate in phlegmonous suppuration is 11.9 per cent and is zero in the abscess form. The prognosis in advanced cases is grave.

The treatment is incision for drainage of the floor of the mouth with the incisional line under the chin. Free pus may not be found; in fact I have seen free pus only twice in eight patients. Tracheotomy must be done at the first sign of glottic edema or dyspnea. This may occur within twenty-four hours of onset.

The major anesthesia problem is how to keep the patient breathing. Adequate respiratory exchange and intake of oxygen are of such major importance in these cases that anesthesia sometimes is primarily a matter of the prevention and treatment of asphyxia.

Several anesthetic agents and methods of anesthesia have been suggested, and each has merit. The anesthetist should certainly administer that with which she is familiar. Jackson[1] advocated local anesthesia only. According to others, a few drops of chloroform or light gas anesthesia is indicated.

In my experience analgesia with nitrous oxide-oxygen has proved satisfactory. One patient out of eight required tracheotomy on the operating table. If the patient is breathing reasonably well, 50 per cent nitrous oxide will obtund pain sufficiently for the necessary incision and drainage to be done.

Within a four day period recently, I had two patients with Ludwig's angina to anesthetize. The following case reports describe the anesthesia and surgical procedure.

1. Jackson, C.; Jackson, C. L., and Coates, G. M.: *The Nose, Throat, and Ear and Their Diseases* (Philadelphia: W. B. Saunders, 1929).
2. Sajous, C. E. de Medicis: *Analytic Cyclopedia of Practical Medicine* (Philadelphia: F. A. Davis Co., 1912-18).
3. Barnhill, F. J., cited by Christopher, F.: *Minor Surgery* (Philadelphia: W. B. Saunders, 1944).

CASE 1.—A man was admitted to the hospital on April 18, 1950, with a history of progressive swelling in the submaxillary area and floor of the mouth since extraction of a lower wisdom tooth on April 14, 1950. On admission he was unable to swallow liquids and had a temperature of 101 F. There were marked swelling and induration in the right submaxillary region of the neck, extending into the submental area across the midline, edema of the floor of the mouth, and severe trismus. The diagnosis was Ludwig's angina with edema of the pharynx and the larynx.

Penicillin, 40,000 units, was given every three hours immediately after hospitalization. There was no improvement in ten hours, and incision and drainage were deemed necessary.

At 9:05 p. m. nitrous oxide-oxygen in a 50-50 per cent mixture was administered. Analgesia was established, and at 9:08 p. m. the head was turned to the left for the surgical procedure. As a result, respiration was shut off, and the head had to be straightened. Respiration resumed spontaneously; it was labored as before but seemingly adequate. At 9:10 p. m. the procedure was repeated and again repeated at 9:18 p. m. This time respiration was not resumed after the head was straightened. Respiratory movements and all other efforts to get oxygen into the lungs were of no avail. Cyanosis progressed to 4 plus, and the pulse became bounding, then weak.

At 9:22 p. m. the trachea was opened. By 9:26 p. m. respirations and color were good, and the operative procedure was continued. At 9:35 p. m. the patient complained of severe pain, and nitrous oxide-oxygen was given in a mixture ranging from 20 to 50 per cent oxygen according to the patient's needs. This was given by holding the casting immediately above the tracheal tube. By 9:50 p. m. anesthesia and the operation were terminated. Respiration and color were good; pulse rate was 120 per minute. The patient was put to bed, and the administration of oxygen continued.

Three days postoperatively the tracheal tube was removed. The patient's mouth could be opened ¼ inch. On April 23 administration of chloromycetin was started and was found to be more effective than penicillin. On April 26 his jaw could be opened ½ inch, and the swelling was less. The patient was discharged from the hospital considerably improved.

CASE 2.—A woman was admitted to the hospital on April 22, 1950, with a history of swelling of the submaxillary area, difficulty in swallowing, and trismus that had been progressive since the extraction of a lower molar tooth eight days previously. On admission she had swelling and hardness of the floor of the month, difficulty in swallowing, and trismus. The diagnosis was Ludwig's angina, with cellulitis of the floor of mouth, mainly on the right. Incision and drainage were thought necessary.

At 7:55 p. m. a mixture of 75 per cent nitrous oxide and 25 per cent oxygen was administered. No respiratory difficulties were encountered. By 8:10 p. m. the operation and anesthesia were terminated. The patient's pulse, respiration, and color were good, and she was returned to bed.

No respiratory difficulties were noted the following day, and on April 24 trismus was reduced. On April 25 there was marked improvement, and the patient was discharged on April 28 in good condition.—HARRIET L. ABERG, Galesburg, Ill.

LEGISLATION

Emanuel Hayt, LL.B.*

Penalties for Failure to Re-register.—The practice of anesthesiology ordinarily is regarded as the practice of medicine. It becomes proper nursing practice by a registered professional nurse only when she is acting under the direction and supervision of a duly licensed physician. She does not differ from the ordinary registered professional nurse in her qualifications, except as a specialist.

All persons who are professional nurses are required to have certain qualifications, and when they practice a nursing specialty it is expected that they will have had the required experience and knowledge. It is a well known fact that professional nurses may be found in various fields, e.g., public health, obstetrics, and surgery.

In some states nurse anesthetists must have a special license. Without such a license in those states they are regarded as illegal practitioners, in the same sense as professional nurses who are unregistered are regarded as illegal practitioners wherever professional nurses must obtain a license or a certificate of registration.

In most states registered professional nurses must maintain this status by re-registration, usually on an annual basis. A digest of the various nursing practice acts and board rules throughout the country, as reported by the American Nurses' Association for the years 1948 and 1949, indicates that the failure to re-register will have the following effects:

Forfeiture of Right to Practice Nursing as a Registered Nurse: Alabama, Colorado, Indiana, Iowa, Kansas, Maine, Massachusetts, Minnesota, Nevada, New Mexico, and Tennessee.

Considered an Illegal Practitioner and Subject to a Hearing: Arkansas, Michigan, Missouri, Montana, New York, South Carolina, Texas, and Vermont.

Cancellation of Registration: District of Columbia, Georgia. Idaho, Kentucky, Nebraska, and Oregon.

Penalty for Failure to Re-register: Florida, Kansas, Louisiana, and North Carolina.

Subject to Suspension of Right to Practice: North Dakota.

Reinstatement on Payment of Renewal Fees: South Dakota, Utah, and Washington.

Private Hospital Is Liable for Injury During Patient's Medical Care.[1]—The courts will hold a private hospital liable for the negligence of a nurse in its employ, even though the injury to the patient arises in the course of medical care rendered under the direction of the patient's own physician. What holds true in the case of an injury caused by an intern applies with greater force to the carelessness of a nurse or other agent or employee of a private hospital. This precise question was answered in a recent case.

An operation to remove a fistula was performed by a surgeon of the patient's own choice in a private hospital. The nature of the operation did not require the attendance or services of a trained nurse. In the course of the operation, while

*Counsel for A.A.N.A.

[1]Bakal v. University Heights Sanatarium, Inc., N.Y.L.J., June 26, 1950, p. 2249, Supreme Court, N. Y. County, Trial Term Part II, Benvenga, J.

the patient was under anesthesia, she sustained severe burns caused by the application of an electric plate connected with an electrocautery knife. The plate was applied to the body of the patient by an unlicensed employee of the hospital, who had received some training as a nurse. She had been instructed by someone connected with the hospital to prepare the patient for the operation. This was done before the surgeon entered the operating room.

The jury found that the injuries and burns were caused by the electric plate, and that the plate was improperly applied to the patient's body by the employee.

There being no evidence of negligence on the part of the physician, the complaint against him was dismissed. The jury found that, under the circumstances of this case, it was not necessary for the doctor to supervise the placing of the electric plate on the patient's body. The jury's award of $7,000 against the hospital was reduced by the court as excessive to $5,000.

A. A. N. A.

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THE NEWS

GERTRUDE L. FIFE TO RECEIVE AWARD OF APPRECIATION

At the annual banquet in Atlantic City, on September 20, Gertrude L. Fife of Cleveland will receive the Annual Award of Appreciation for her many years of outstanding service to the American Association of Nurse Anesthetists.

Mrs. Fife has served as the Association's treasurer since 1935 and was also the Association's second president and first editor. She is a graduate of the Fanny Allen Hospital School of Nursing, Winooski, Vt., and the Lakeside Hospital School of Anesthesia, Cleveland. From 1934 to 1947 she was director of the University Hospitals School of Anesthesia in Cleveland. She has been described by the *American Journal of Nursing* as "the most widely known nurse anesthetist in this country."

Invitations have been extended to each state president to sit at the banquet table.

Quiz Program

On the afternoon of September 21 the Publications Committee is to conduct a quiz program for which prizes are being donated by the state associations. Acting as judges will be the members of the committee: Opal Schram, chairman, Harriet Aberg, Agnes Lange, Betty E. Lank, and Ruth Schierman. Questions will be taken from the Journal, May 1949 through August 1950, and the A. A. N. A. News Bulletin, June 1949 through September 1950. All members of the audience will be invited to participate.

Business Session

Accreditation of schools of anesthesia for nurses is the major issue before the members at the business session on September 19. Delegates will be asked to go on record as approving the principles of accreditation as presented by Helen Lamb, chairman of the Advisory Committee. An increase in dues to cover the cost of accreditation will also be voted on, as well as a two year assessment to launch the program. The revision to the *Bylaws* pertaining to the increase in dues and the assessment is published on page 207 of this Journal.

The election of officers is scheduled for 11:30 a. m. September 19. Candidates and their qualifications are published on page 199 of this Journal.

NOMINATIONS FOR OFFICE AMERICAN ASSOCIATION OF NURSE ANESTHETISTS

1950-51

At the Seventeenth Annual Meeting in Atlantic City, September 18-21, the Nominating Committee will present a ballot of candidates for office that will include the following members who have qualified and have given their consent to serve.

PRESIDENT

Verna E. Bean (Lexington, Ky.): Graduate of St. Elizabeth's Hospital School of Nursing, Boston; graduate of Long Island College Hospital School of Anesthesia, Brooklyn; Army Nurse Corps, three and one-half years; member of A.A.N.A. in good standing since 1933; former president, New York Association of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1947-49; 2nd vice president, A.A.N.A.; chairman, Public Relations Committee, A.A.N.A.

1ST VICE PRESIDENT

Josephine Bunch (Shriners' Hospital for Crippled Children, Portland, Ore.): Graduate of Sacred Heart Hospital School of Nursing, Spokane, Wash.; graduate of St. Vincent's Hospital School of Anesthesia, Portland, Ore.; member of A.A.N.A. in good standing since 1936; former president, Oregon Association of Nurse Anesthetists; former chairman, Western States Assembly of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1948-50; chairman, Personnel Practices Committee, A.A.N.A.

2ND VICE PRESIDENT

Minnie V. Haas (St. Joseph's Hospital, Fort Worth, Tex.): Graduate of Methodist Deaconess Hospital School of Nursing, Rapid City, S. Dak.; graduate of Lakeside Hospital School of Anesthesia, Cleveland; member of A.A.N.A. in good standing since 1934; former president, Texas Association of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1948-50.

TREASURER

Agnes M. Lange (Ravenswood Hospital, Chicago): Graduate of Mt. Carmel Hospital School of Nursing, Columbus, Ohio; graduate of St. Joseph's Hospital School of Anesthesia, Chicago; member of A.A.N.A. in good standing since 1935; secretary-treasurer, Indiana Association of Nurse Anesthetists, 1937-50; member, Tri-State Assembly Board of Trustees, 1940-50; member, Publications Committee, A.A.N.A.

TRUSTEES

Rosella J. Crotty (Luther Hospital, Eau Claire, Wis.): Graduate of St. Mary's Hospital School of Nursing, Duluth; graduate of St. Mary's Hospital School of Anesthesia, Duluth; member of A.A.N.A. in good standing since 1938; president, Wisconsin Association of Nurse Anesthetists; member, Nominating Committee, A.A.N.A.

Harriet L. Aberg (Cottage Hospital, Galesburg, Ill.): Graduate of St. Luke's Hospital School of Nursing, St. Paul; graduate of Minneapolis General Hospital School of Anesthesia, Minneapolis; member of A.A.N.A. in good standing since 1934; former vice president, New Jersey Association of Nurse Anesthetists; member, Publications and Educational Exhibits Committees, A.A.N.A.; A.A.N.A. representative on Hospital Operating Rooms Committee.

Esther E. Edwards (Wausau Memorial Hospital, Wausau, Wis.): Graduate of Wesley Memorial Hospital School of Nursing, Chicago; graduate of Ravenswood Hospital School of Anesthesia, Chicago; member of A.A.N.A. in good standing since 1938; former president, Eighth District, Wisconsin State Nurses' Association; former president, Wisconsin Association of Nurse Anesthetists.

Martha S. Jackson (Municipal Hospital, Tampa, Fla.): Graduate of Macon Hospital School of Nursing, Macon, Ga.; graduate of Long Island College Hospital School of Anesthesia, Brooklyn; member of A.A.N.A. in good standing since 1943; former president, Florida Association of Nurse Anesthetists; chairman, Southeastern Assembly of Nurse Anesthetists; chairman, Nominating Committee, A.A.N.A.

Edna Peterson (St. Francis Hospital, San Francisco): Graduate of St. Joseph's Hospital School of Nursing, Vancouver, Wash.; graduate of Sacred Heart Hospital School of Anesthesia, Spokane, Wash.; member of A.A.N.A. in good standing since 1941; former president, California Association of Nurse Anesthetists; former chairman, Western States Assembly of Nurse Anesthetists; chairman, Nominating Committee, A.A.N.A., 1948-49.

Hazel J. Peterson (Deaconess Hospital, Minneapolis): Graduate of University of Minnesota School of Nursing, Minneapolis; graduate of St. Joseph's Hospital School of Anesthesia, Chicago; charter member, A.A.N.A.; former president, Minnesota Association of Nurse Anesthetists; former secretary, Minnesota Association of Nurse Anesthetists; chairman, Revisions Committee, A.A.N.A.

Verna M. Rice (Mobile, Ala.): Graduate of the Sarah Leigh Hospital School of Nursing, Norfolk, Va.; graduate of the Lakeside Hospital School of Anesthesia, Cleveland; charter member of A. A. N. A.; former 1st vice president, A. A. N. A.; former member, Board of Trustees, A. A. N. A.; former chairman, Revisions Committee, A. A. N. A.; former chairman, Trust Fund Committee, A. A. N. A.

SEVENTEENTH ANNUAL CONVENTION

AMERICAN ASSOCIATION OF

NURSE ANESTHETISTS

September 18-21, 1950

ATLANTIC CITY

Hotel Headquarters—Ritz-Carlton

All General Sessions and the Business Session will be held in Convention Hall, Room A.

PROGRAM

Sunday, September 17

10:00 A.M.-6:00 P.M.— **Registration**
A.H.A. Headquarters—Hotel Traymore

9:00 A.M.— **Assembly of Directors of Schools of Anesthesia**
Ritz-Carlton Hotel

Rosella Crotty, R.N.
Director, School of Anesthesia
Luther Hospital, Eau Claire, Wis.
Presiding Officer

Address of Welcome
Myra Van Arsdale, R.N.
President, A.A.N.A.

Test Construction as Used by Examination Committee
Janet McMahon, R.N.
Educational Director, School of Anesthesia
University Hospitals, Cleveland

Accreditation of Schools
Helen Lamb, R.N.
Director, School of Anesthesia
Barnes Hospital, St. Louis

2:00 P.M.— Lillian G. Baird, R.N.
Director, School of Anesthesia
University Hospital, Ann Arbor
Presiding Officer

Selection of Students for Schools of Anesthesia
Miriam G. Shupp, R. N.
Director, School of Anesthesia
University Hospitals, Cleveland

Open Discussion

Monday, September 18

8:00 A.M.—**Registration**

Registration Desk—Convention Hall

9:00 A.M.—**Assembly of Directors of Schools of Anesthesia**

Convention Hall, Room A

Forum: Teaching Methods

Helen Vos, R.N.
Director, School of Anesthesia
Hurley Hospital, Flint, Mich.
Moderator

Mary A. Costello, R.N.
Cincinnati General Hospital, Cincinnati

Margherita Powers, R.N.
Johns Hopkins Hospital, Baltimore

Opal Schram, R.N.
Wesley Memorial Hospital, Chicago

Edith McGinley, R.N.
Ravenswood Hospital, Chicago

Sister Seraphia, R.N.
St. John's Hospital, Springfield, Ill.

2:00 P.M.— **General Session**

Convention Hall, Room A

Myra Van Arsdale, R.N.
President, A.A.N.A.
Presiding Officer

Invocation

Sister Mary Manuela
Lodi, N. J.

Address of Welcome

Myra Van Arsdale, R.N.
President, A.A.N.A.

Address of Welcome from A.H.A.

Charles Wilinskey, M.D.
President-elect, A.H.A.
Beth Israel Hospital, Boston

2:30 P.M.— Eunice Poss, R.N.
President, Virginia Association of Nurse Anesthetists
Presiding Officer

Anesthetic Problems in Plastic Surgery
P. C. Iverson, M.D.
Instructor in Plastic Surgery, Graduate School of Medicine
University of Pennsylvania, Philadelphia
and
Olga Schweizer, M.D.
Director of Anesthesiology
Memorial Hospital, New York City

4:00 P.M.—**Use of Cathode Ray Scanners for Recording Physiologic Data during Operative Procedure**
H. A. Zimmerman, M.D.
Director, Cardiovascular Laboratory
Cleveland City Hospital, Cleveland

7:00 P.M.—**Southern States Dinner**
Ritz-Carlton Hotel

Tuesday, September 19

9:00 A.M.— **Business Session**
Convention Hall, Room A

Myra Van Arsdale, R.N.
President, A.A.N.A.
Presiding Officer

Call to Order
Reading of Minutes
Reports of Officers
Report of Nominating Committee
11:00 A.M.—**Election of Officers**

2:00 P.M.— **Business Session**
Convention Hall, Room A

Myra Van Arsdale, R.N.
President, A.A.N.A.
Presiding Officer

Socialized Medicine: The Stalking Horse of Communism
F. F. Borzell, M.D.
Speaker, House of Delegates, A.M.A.
Philadelphia

Reports of Standing Committees
Reports of Special Committees
Unfinished Business
New Business

4:00 P.M.— **Council Session**
Convention Hall, Room A

Florence A. McQuillen, R.N.
Executive Director, A.A.N.A.
Presiding Officer

Wednesday, September 20

9:00 A.M.— **Joint Session with A.H.A.**
Convention Hall, Room A

F. R. Bradley, M.D.
Director, Barnes Hospital, St. Louis
Presiding Officer

Analysis of Hospital Anesthesia Service

Introduction to the Subject
Vane M. Hoge, M.D.
Assistant Surgeon General, Bureau of Medical Service
Public Health Service, Washington, D. C.

As Viewed by the Nurse Anesthetist
Verna E. Bean, R.N.
2nd Vice President, A.A.N.A.
Lexington, Ky.

As Viewed by the Medical Anesthesiologist
Donald E. Hale, M.D.
Anesthesiologist
Cleveland Clinic Hospital, Cleveland

As Viewed by the Surgeon
Adolph Walkling, M.D.
Associate Professor of Surgery
Jefferson Medical College, Philadelphia

Discussion

2:00 P.M.— **General Session**
Convention Hall, Room A

Emma Miller, R.N.
President, Pennsylvania Association of Nurse Anesthetists
Presiding Officer

Complications of Spinal Anesthesia
C. L. Hebert, M.D.
Anesthesiologist
U. S. Marine Hospital, Staten Island, N .Y.

3:00 P.M.— **Evaluation of Pentothal Sodium in Anesthesia**
Vincent J. Collins, M.D.
Anesthesiologist
St. Vincent's Hospital, New York City

4:00 P.M.— **Clinical Uses of Procaine Administered Intravenously**
D. J. Graubard, M.D.
Adjunct Visiting Surgeon
Beth David Hospital, New York City

7:30 P.M.— **Banquet**
Ritz-Carlton Hotel—Trellis Room
Myra Van Arsdale, R.N.
President, A.A.N.A.
Presiding Officer

Invocation

Presentation of A.A.N.A. Award of Appreciation
Gertrude L. Fife, R.N., *Receiving*
Past-president and Treasurer, A.A.N.A.
Cleveland Heights, Ohio

Nurses Should Talk Well, Too
Richard C. Reager
Chairman, Speech and Debate Department
Rutgers University, New Brunswick, N. J.

Thursday, September 21

9:00 A.M.— **General Session**
Convention Hall, Room A
Helen Roe, R.N.
President, Indiana Association of Nurse Anesthetists
Presiding Officer

10:00 A.M.—**Subject and speaker to be announced**

11:00 A.M.—**A School for Nurse Anesthetists Can Be Successful**
W. O. Bohman, Administrator
Norwegian-American Hospital, Chicago

2:00 P.M.— **General Session**
Convention Hall, Room A
Madelyn Burns, R.N.
President, Maryland Association of Nurse Anesthetists
Presiding Officer

Prophylactic and Therapeutic Use of Vasopressors in Anesthesia
R. A. Hingson, M.D.
The Johns Hopkins Hospital, Baltimore

2:45 P.M.— **Quiz Program**
Conducted by the A.A.N.A. Publications Committee
Gertrude L. Fife, R.N.
Associate Editor, *Journal A.A.N.A.*
Mistress of Ceremonies

4:30 P.M.— **Unfinished Business**

Adjournment

PROPOSED REVISION TO BYLAWS

The revision to the *Bylaws of the American Association of Nurse Anesthetists* as published herein will be presented to the members at the business session of the Seventeenth Annual Meeting on September 19, 1950, in Atlantic City.

Revisions Committee
PALMA ANDERSON
PAULINE HENRY
HAZEL PETERSON, Chairman

PRESENT BYLAW

ARTICLE IX

DUES

Section 1.—Amount

Annual dues are determined according to the following classification and are payable in advance on September 1.

Classification	Dues	Retained by Local Association
Active Members	$12.00	$4.50
Active Members pro rata payment (see note)	6.00	2.25
Inactive Members	5.00	2.00
Contributing Members	15.00	4.50
Sustaining Members	25.00	5.00
Institutional Members	25.00	5.00

Note: For members accepted after March 1.

PROPOSED REVISION

ARTICLE IX

DUES

Section 1.—Amount

A. Annual dues are determined according to the following classification and are payable in advance on September 1.

Classification	Dues	Retained by Local Association
Active Members	$15.00	
Active Members pro rata payment	$ 7.50	

B. There shall be an assessment of $5.00 per Active Member to be paid in addition to and at the time of the payment of the dues for each of the fiscal years, 1951 and 1952, the funds from the assessment to be reserved for the establishment and maintenance of a program for the approval of schools of anesthesia for nurses.

PEOPLE AND EVENTS

At the annual meeting of the **Iowa** association on April 21, the members voted to go on record as opposed to compulsory health insurance. Zipora Braunschweig was elected secretary, and Lucille V. Girton, trustee.

At a meeting of the **North Carolina** association in Charleston, S. C., May 11-12, officers elected were: Lottie Edwards, president; Sophia Chapman, vice president; Marie Guthrie, secretary; Julia B. Henry, treasurer.

The annual business meeting of the **Maryland** association was held at the Hotel Emerson, Baltimore, on May 13. Officers elected were: Madelyn Burns, president; Margherita Powers, vice president; Grace Smith, treasurer; Mary Elizabeth Long, secretary.

At the **Middle Atlantic** Assembly of Nurse Anesthetists, convening in Buffalo, May 24-26, officers elected were: Rose Donovan, chairman; Marie A. Bader, 1st vice chiarman; Janet Cabedo, 2nd vice chairman; Jean Zanger, secretary-treasurer.

Officers for the **Western States** Section of Nurse Anesthetists for 1951 are Rose Gish, chairman; Jeanne Fagan, vice chairman; Marie Hebert, secretary-treasurer; Ruth Walther, trustee, and Mary Fleming, alternate.

DEATHS

Mrs. Sybil G. Stitt, a member of the Illinois association, died after a long illness at her home in Kennett, Mo., on May 14, 1950. She was a graduate of the Grace Hospital School of Anesthesia, Detroit.

Edith H. Jones, a member of the California association, died on May 30, 1950, at St. Francis Hospital, San Francisco, where she had been chief anesthetist for fourteen years. She received her training in anesthesia at Lakeside Hospital, Cleveland.

ABSTRACTS

Hebert, C. L.; Tetirick, C. E., and Ziemba, J. F.: Complications of spinal anesthesia: an evaluation of the complications encountered in 5,763 consecutive spinal anesthesias. J. A. M. A. **142**:551-557, Feb. 25, 1950.

"Over a period of several decades spinal anesthesia has proved to be a satisfactory method in the United States Marine Hospitals. In this paper we present the complications that we have observed in a series of 5,763 consecutive single dose administrations of spinal anesthesia during a thirty-nine month period beginning Jan. 1946 at one of these hospitals. . . . Although we employ spinal anesthesia in all types of surgery below the diaphragm, as a rule we tend to use it chiefly for operations confined to the lower part of the abdomen or the abdominal wall and for procedures on the lower extremities. . . . The majority of the patients in this series were relatively healthy male merchant seamen or civil service workers engaged in physical labor. For the most part they were in the fourth and fifth decades of life. . . .

"Spinal anesthesia was administered by an experienced physician anesthetist or by an intern or resident under his direct supervision. A standard technic of lumbar puncture, essentially that of Lundy utilizing an introducer, was adhered to in practically every case. . . . After spinal anesthesia was produced, the patient was observed and managed by a physician or nurse anesthetist, with an experienced physician anesthetist constantly available for consultation. Supportive measures were instituted on the slightest indication. Spinal anesthesia was combined with some form of general anesthesia whenever the patient was unduly apprehensive, the spinal nerve block was inadequate or the operation was prolonged beyond the duration of the block. . . . Information concerning postanesthetic complications was obtained by direct questioning of the patient and ward personnel by an anesthetist forty-eight hours after the operation and by a check with the resident ward surgeon two weeks postoperatively. Complications which were present for more than two weeks were followed by reviewing the patient's hospital record after his discharge. . . . The complications occurring immediately after the establishment of spinal anesthesia are directly associated with the physiologic and psychologic disturbances produced by the method. Most of these phenomena are relatively minor and are easily corrected if recognized early. . . . Only a small proportion of our patients (1.8 per cent) exhibited nervousness and apprehension. . . . Inadequate or ill timed premedication was usually noted in these patients. Supplemental medication . . . readily corrected the situation. . . . Nausea and vomiting appeared in 6.3 per cent of our patients. A number of factors seem to contribute to the production of these symptoms.

"Sudden changes in position, such as are necessary in giving spinal anesthesia, are known to elicit these symptoms in patients premedicated with morphine, as were all the patients in the series. Also, it is a common observation that patients with spinal anesthesia are likely to respond to postural changes with these symptoms. . . . The emotional factor must also be considered. Patients are apt to respond to psychic trauma with nausea and vomiting. Proper premedi-

cation is most helpful in preventing, and supplemental medication in controlling, this factor. Increased muscular tone of the gastrointestinal tract coupled with relaxation of the pyloric sphincter produced by splanchnic nerve block favors the production of nausea and vomiting. On the other hand, incomplete splanchnic nerve block favors reflex nausea and vomiting produced by visceral and mesenteric traction. . . . Fourteen per cent of the patients exhibiting nausea and vomiting had a decided blood pressure fall. The administration of oxygen and the restoration of the blood pressure to normal levels by usual methods helped to alleviate the condition. . . . The level of anesthesia was unnecessarily high for the contemplated surgical procedure and was associated with circulatory and respiratory embarrassment of the patient in 5.7 per cent of the anesthesias in this series. Asymptomatic high spinal anesthesias were not recorded as complications. The height of anesthesia to be expected in a given patient even with a standard technic is not always predictable. The site of injection, as long as it is in the lumbar region, plays a minor role. . . . The level of anesthesia must be carefully ascertained in each case after waiting long enough for the drug to become 'fixed.' Death can occur from high spinal anesthesia, when the level has not been determined or has been masked by supplemental medication and when proper supportive measures have not been instituted. In our experience the incidence of inadvertent high spinal anesthesia was considerably less when hyperbaric tetracaine hydrochloride (pontocaine hydrochloride) dextrose-ephedrine was used as compared to piperocaine hydrochloride (metycaine hydrochloride) or procaine hydrochloride given in isobaric solution. . . .

"Cardiovascular depression is the most important complication seen during spinal anesthesia from the standpoint of frequency and potential harm to the patient. It is to be anticipated in every case; hence, a vasoconstrictor drug is usually given before the administration of the spinal anesthesia. All our patients received 25 mg. ephedrine intramuscularly immediately before the subarachnoid injection was performed. Admittedly, it would be preferable to have given this drug at least fifteen minutes earlier. Moderate blood pressure fall, which we defined as a drop of the systolic pressure from normal levels to between 80 and 90 mm. of mercury, or a fall in the systolic pressure of the magnitude of 30 to 40 mm. of mercury, occurred in 7 per cent of the cases. Decided blood pressure fall, which we defined as a drop of the systolic pressure below 80 mm. of mercury, or a fall of the systolic pressure of more than 40 mm. of mercury, occurred in 4.2 per cent. . . . It is our impression that the more rapidly acting drugs procaine and piperocaine, are more apt to cause blood pressure fall than is the slower-acting drug tetracaine, even though the ultimate level of anesthesia produced by both groups of agents is the same. . . . Regardless of whether hypotension is due primarily to diminished cardiac output or to increased capacity of the vascular bed through arteriolar dilatation, the degree of hypotension is proportional to the number of spinal nerve roots blocked and the condition is usually corrected by the administration of ephedrine or related drugs which have a tonic effect on both the heart and the sympathetic myoneural junctions of the arterioles.

"In the management of moderate

hypotension it was often found beneficial to administer rapidly a small quantity (up to 500 cc.) of dextrose-saline solution intravenously instead of administering vasoconstrictors. Oxygen was given routinely, and often it was the only supportive measure used. Obviously, one should avoid spinal in patients with coronary artery disease in whom a sudden drop in the diastolic pressure for even a short period may be disastrous. . . . Spinal anesthesia which is insufficient to permit the operation to begin or which fails to produce anesthesia for the usually expected period is a complication which is difficult to prevent. . . . We encountered this phenomenon in 98 instances (1.7 per cent). For purposes of this study we recorded anesthesias with piperocaine and procaine as being inadequate if their duration was less than sixty minutes. Tetracaine anesthesia was considered inadequate if the duration was less than ninety minutes. . . . When the foregoing definitions are applied, tetracaine gave incomplete or inadequate anesthesia in 3.5 per cent of the instances in which it was used as compared with 1 per cent for piperocaine. This may be explained partially by the fact that we were using the relatively small doses of tetracaine, combined with ephedrine and dextrose, advocated by Potter and Whitacre. Failure to obtain spinal anesthesia after apparent successful intrathecal injection of an agent is not uncommon. We experienced this result in 41 instances (0.7 per cent). Tetracaine administration was followed by the highest proportion of failures, 1.5 per cent, as compared with piperocaine, 0.4 per cent.

"Failure to place all or part of the anesthetic agent into the subarachnoid space is credited with being responsible for 99 per cent of failures in spinal anesthesia by Cullen. High alkalinity of the spinal fluid causing precipitation of the alkaloid anesthetic base has been reported as the cause of failure by Cohen and Knight. The possibility that the ampul contents are not as stated on the label must also be considered. In 20 instances we repeated the spinal injection. The second administration was unsuccessful in 6 patients. Miscellaneous rare accidents such as needle breakage, toxic reactions to drugs and deaths directly attributable to spinal anesthesia were not encountered in this series. . . . Complications occurring in the postoperative period which may be attributed to spinal anesthesia may be due to the changes produced during the anesthetic period or could be due to the introduction of the spinal needle or certain drugs into the subarachnoid space. Frequently the effects of preexisting disease, drugs used in premedication or supplemental anesthesia, position of the patient during the operation and surgical manipulations may be wholly or partially responsible for some of the so-called 'post-spinal-anesthesia complications.' In presenting the following complications we have attempted to screen out those conditions which obviously were due to these extraneous factors. . . . Postoperative nausea and vomiting was reported in 2 per cent of our patients and was of not more than twenty-four hours' duration in most instances. Hiccup was reported in 8 patients of the entire series after operations involving the lower portion of the abdomen; in 3 patients it persisted for one week.

"Abdominal distention occurred in only 10 patients postoperatively.

Several of these patients had had a protruded intervertebral disk removed. We are unable to relate most of these complications to the method of anesthesia. Their management depends on the individual case. . . . Respiratory complications occurred in 1.63 per cent of patients. . . . In this series 106 patients (1.83 per cent) complained of backache, which persisted for more than two days in only 5 patients. In 10 instances multiple insertions of the introducer or spinal needle were recorded. This incidence of multiple puncture does not differ significantly from that in the series as a whole. Therefore we do not believe that it played an important part in the development of backache. Seemingly, relaxation of the lumbar muscles during anesthesia is a more important factor. The placement of small pillows under the lumbar curve and beneath the knees has been suggested. We have not done this routinely. . . . Urinary retention of some degree occurred in 177 patients, an incidence of 3 per cent. . . . Headache was reported by 371 patients (6.43 per cent). . . . Two headaches were associated with stiffness of the neck muscles. These followed repeated spinal punctures with bloody taps. Only 15 headaches were of more than one week's duration. . . .

"We kept a record of the number of times the introducer and needle were inserted in the back and spinal structures. Twenty per cent of the patients having headache had two or more insertions of the spinal needle, whereas the incidence of multiple punctures in the entire series was approximately 10 per cent. Thus, multiple insertions of the spinal needle may be a factor in the causation of headache. Headache due to cerebrospinal fluid hypotension is aggravated by the upright position and relieved by recumbency. However, early ambulation which has been in vogue at our hospital for the past few years has not seemed to increase the incidence of headache. . . . Severe neurologic complications did not follow the administration of the 5,763 spinal anesthesias in this series. In 2 patients unilateral ulnar neuropathy developed, presumably from contact of their elbows against the unpadded edge of the operating table. Four patients had stiffness and soreness of cervical muscles up to five days postoperatively. Two of these had associated headache; multiple punctures and bloody taps had been obtained on both. None of these patients had elevation of temperature above 100 F. It is presumed that they manifested a mild aseptic or hemorrhagic meningitis. Six patients had evidence of involvement of the cauda equina. One had a unilateral foot drop which persisted for three weeks, after which improvement occurred. Another had weakness of one leg for several days with no residual disability at the time of discharge. The other 4 patients had transient pain or paresthesia in one leg, the longest duration of which was twelve days. There was no difficulty in performing lumbar puncture in any of these cases. Tetracaine and piperocaine were equally culpable. . . . The possibility of late neurologic sequelae after spinal anesthesia must always be considered. No such instances have come to our attention."

BOOK REVIEWS

PSYCHOLOGY AND THE NURSE. By Frank J. O'Hara, C.S.D., Ph.D., Dean of the School of Science, Stonehill College, North Easton, Mass. Ed. 3. Cloth. 253 pages, 18 figures. Philadelphia: W. B. Saunders Co., 1949. $2.75.

As in the two previous editions, the author's purpose in this third edition is to present the principles of general psychology clearly and concisely to freshmen students in schools of nursing. This he does in an interesting manner. Instead of the usual rather dry and uninteresting presentation, we found this book enjoyable reading.

While it is primarily designed as a text for the student nurse, the nurse anesthetist will find many principles that may be applied to her special field of work and that will aid her in her dealings with her fellow workers and in the handling of the various diverse personalities that her patients present. This is especially true of the chapter on "Personality." At the end of each chapter there are a concise summary and a list of suggested questions for review. Indexed.—AGNES LANGE, Ravenswood Hospital, Chicago.

CLINICAL USES OF INTRAVENOUS PROCAINE. By David J. Graubard, M.D., Assistant Visiting Surgeon, Cumberland Hospital; Assistant Visiting Orthopedist, Kingston Avenue Hospital, Brooklyn, N. Y., and Milton C. Peterson, M.D., Visiting Anesthesiologist, Research Hospital, Kansas City, Mo. Cloth. 104 pages. Illustrated. Springfield, Ill.: Charles C Thomas, Publisher, 1950. $2.25.

This monograph in American Lectures in Anesthesiology is publication 73, American Lecture Series. There are chapters on history, chemistry and pharmacology, intravenous local anesthesia, treatment of pruritus, serum sickness, burns and postoperative pain, arrhythmias during anesthesia, pain in traumatic and inflammatory conditions, acute anterior poliomyelitis, and other conditions. Some of the information is in the nature of preliminary reports on the use of procaine for certain conditions. Tables, charts, graphs, footnotes, pictures, and case reports are used to present much information in a concise manner. Each section is followed by a list of references. Indexed.

FUNDAMENTALS OF ANESTHESIOLOGY FOR THE NURSE ANESTHETIST. By Sr. M. Bonosa, Director of the Sacred Heart Hospital School of Anesthesiology, Allentown, Pa. Fiberboard. 225 pages. Allentown, Pa.: Sacred Heart Hospital School of Anesthesiology, 1950. $3.75.

In this mimeographed handbook the teaching staff of the Sacred Heart Hospital School of Anesthesiology has collected the notes that have been used in presenting classwork to the students. The material is a summary of material collected from articles and journals to supplement available textbooks. The author does not suggest that the notes contain all that the nurse anesthetist should know but presents them as a summary of the fundamentals of anesthesia. For greater detail the student is urged to consult various textbooks. To aid in this, each chapter is followed by a pertinent bibliography of source material.

The notes are divided into six major sections: (I) Anesthesia in General; (II) Anatomy and Physiology in Relation to Anesthesia; (III) Gas Therapy; (IV) Pharmacology in Relation to Anesthesia; (V) Chemistry and Biochemistry in Relation to Anesthesia; (VI)

Miscellaneous Subjects. The handbook is subdivided into thirty-four chapters.

The author is to be commended on this collection of notes that represents a prodigious amount of painstaking labor.

Nursing History in Brief. By Minnie Goodnow, R.N. Ed. 3. Cloth. 274 pages, 128 illustrations. Philadelphia: W. B. Saunders Co., 1950.

In this brief survey of the history of nursing, the author has attempted to show not only the important events but also the stories of many persons who participated in those events. A chronology for general history and one for nursing history precede the text. The only reference to anesthesia is quoted in full:

> The Mayo brothers of Rochester, Minnesota, were probably the first to use nurses as anesthetists; and Dr. George Crile, of Cleveland, was the first to train them in this work. The first trained nurse anesthetist was Alice McGaw [sic], of Chicago.
>
> There has been much controversy among doctors about nurse anesthetists, many of them holding that it was practicing medicine; some interesting legal decisions have been made in the matter. Since there are few doctors interested in or trained for anesthesia, nurse anesthetists in great numbers are required.
>
> *The National Society of Nurse Anesthetists* [sic] has more than 3000 members, all of whom were required to pass a special examination.

Groves and Brickdale's Text-Book for Nurses. Edited by J. A. Nixon, C.M.G., M.D. (Cantab.), F.R.C.P. (Lond.) Emeritus Professor of Medicine, University of Bristol, and Sir Cecil Wakeley, K.B.E., C.B., D.Sc., F.R.C.S., F.R.S.E., Fellow of King's College, London. Cloth. 698 pages, 248 illustrations. New York: Oxford University Press, 1948. $9.25.

This is a combination textbook very different from most of those used in the United States. It covers anatomy, physiology, surgery, medicine, communicable diseases, hygiene, dietetics, a little lab work, and some good advice. Because so much is crowded into it, its worth is questionable. If used only as an outline, it might serve a purpose.

The reason for the inclusion of so much material is stated in the preface to the first edition. "The ordinary student's text-books are not suitable for nurses, because they are too long and expensive, and they presuppose a practical knowledge of laboratory and post-mortem work. On the other hand, the various nursing manuals do not embrace the four subjects of Anatomy, Physiology, Medicine, and Surgery sufficiently comprehensively to form either an examination standard or a book of reference."

According to the authors' nomenclature, local and general are two *grades* of anesthesia. Topical application is *permeation*. On page 263 the description of giving nitrous oxide suggests that it is given only to out-patients by a foot-feed machine. It is also stated that "the routine use of chloroform has been given up, . . ." For the treatment of postanesthesia nausea, the suggested treatment is iced champagne.

The use of heat is emphasized in the treatment of postanesthesia patients. The only precaution for aspiration recommended is turning the head of the patient to the side and keeping it low. Suction is never mentioned, only swabbing of the throat with gauze sponges. Treatment for shock undoubtedly includes many things, but bandaging the "limbs" and adrenalin-saline solution given intravenously are those mentioned.

Some of the illustrations are very good, while others are obviously outdated. Indexed. — Harriet L. Aberg, Cottage Hospital, Galesburg, Ill.

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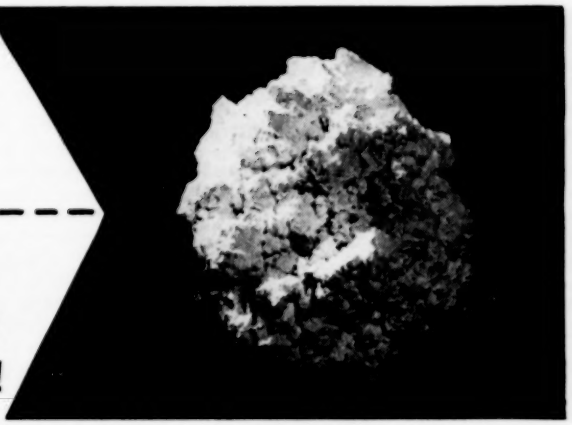

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(*Continued on Page* 218)

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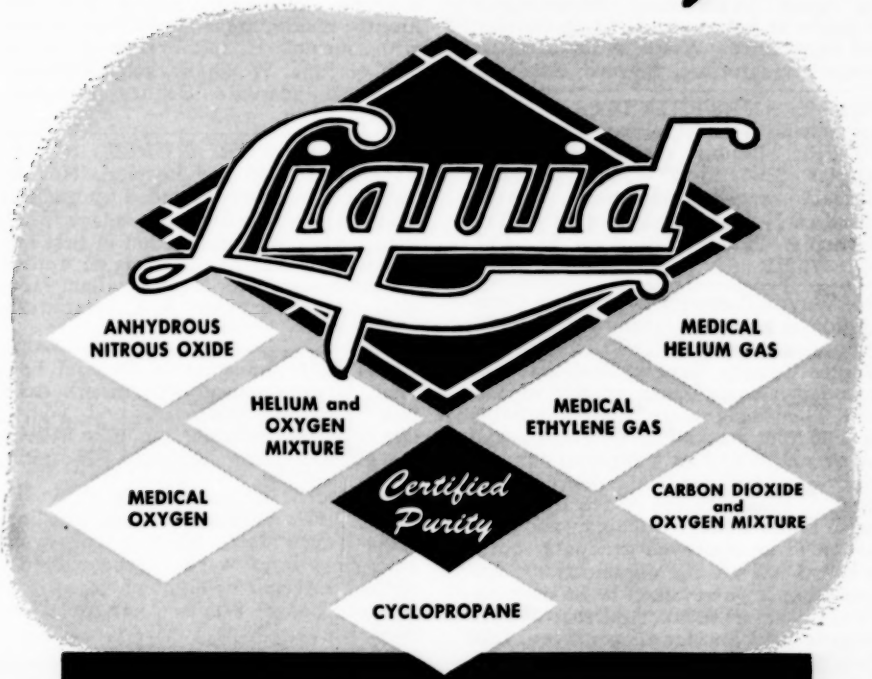

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INDEX TO ADVERTISERS

Bilhuber-Knoll Corp. 220
Dewey and Almy Chemical Co. 215
Edison 148
E & J Manufacturing Co. 143
Liquid Carbonic Corporation 217
Mallinckrodt Chemical Works 219
Merck and Company, Inc. 142
McKesson Appliance Company 145, 147
Ohio Chemical and Mfg. Company ..... BC
Puritan Compressed Gas Corp. 1FC
Linde Air Products Co., Union Carbide and Carbon Corp. 1BC
St.Paul-Mercury Indemnity Co. 197
E. R. Squibb & Sons 141
Classified Advertisements 216

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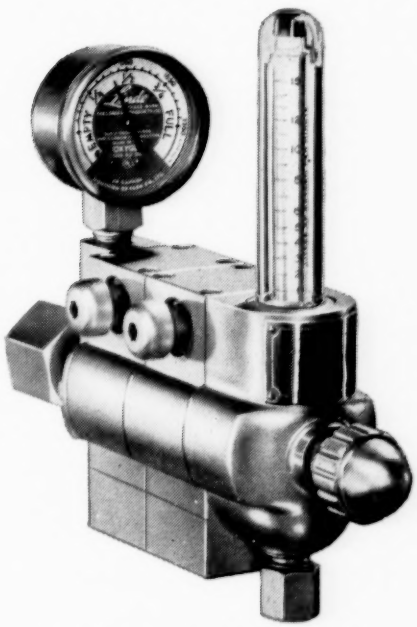

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